Dissertation on

ASSOCIATION OF SYSTEMIC FACTORS IN PRIMARY OPEN ANGLE GLAUCOMA

Submitted in partial fulfillment of requirements of

M.S. OPHTHALMOLOGY

BRANCH - III

REGIONAL INSTITUTE OF OPHTHALMOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI- 600 003

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

MAY 2018

CERTIFICATE

This is to certify that this dissertation entitled **"ASSOCIATION OF SYSTEMIC FACTORS IN PRIMARY OPEN ANGLE GLAUCOMA"** is a bonafide record of the research work done by **Dr. K.BHAGYALAKSHMI,** post graduate in Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Madras Medical College and Government General Hospital, Chennai-03, in partial fulfillment of the regulations laid down by The Tamil Nadu Dr. M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2015-2018.

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INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

To
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Dear Dr.Bhagyalakshi.K.,

The Institutional Ethics Committee has considered your request and approved your study titled **"ASSOCIATION OF SYSTEMIC FACTORS IN PRIMARY OPEN ANGLE, GLAUCOMA" - NO.15012017 (III).**

The following members of Ethics Committee were present in the meeting hold on **24.01.2017** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.M.K.Muralidharan,MS.,M.Ch.,Dean, MMC,Ch-3	:Deputy Chairperson
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12.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

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I hereby declare that this dissertation entitled **"ASSOCIATION OF SYSTEMIC FACTORS IN PRIMARY OPEN ANGLE GLAUCOMA"** is a bonafide and genuine research work carried out by me under the guidance of Prof.Dr.P.S.Maheswari.

DATE : **DR.K.BHAGYALAKSHMI**

PLACE:

CONTENTS

INTRODUCTION

Glaucoma is a blinding cause of optic neuropathy, chronic in nature, progressive disease, characterized by increased intraocular pressure which is the treatable one, optic nerve head changes and corresponding visual field defects in automated perimetry. It occurs due to retinal ganglion cell death. It is also associated with retinal nerve fibre layer defects, disc hemorrhages and peripapillary atrophy.

AQUEOUS HUMOR DYNAMICS:

Aqueous humor is a colorless fluid secreted from ciliary process into the posterior chamber, passes via pupil, enters the anterior chamber.

CILIARY PROCESS:

These are finger like projections projecting from pars plicata. They are 70 in number. The ultra-structure consists of

- central capillaries which has fenestrated endothelium,
- stromal part,
- two layered epithelium, both layers are connected with each other by apical apposition[1].

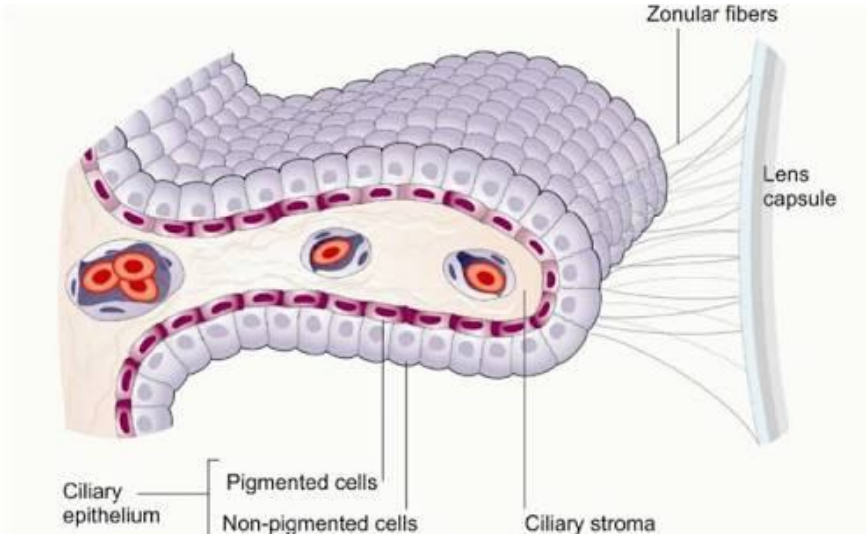

Figure 1 : Ciliary process

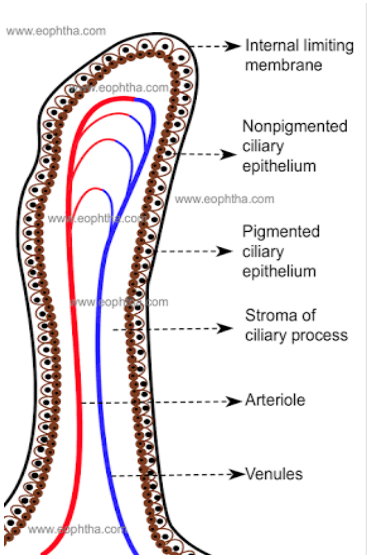

Figure 2 : Anatomical layers of ciliary process

From the anterior chamber it is drained via two pathways namely,

1.Trabecular pathway

2. Uveoscleral pathway.

1. TRABECULAR OUTFLOW:

It is the main outlet pathway, 80%-90% of aqueous humor is draining out by this pathway. Through the trabecular meshwork it enters schlemm's canal, and drained into collector channels and episcleral veins.

2. UVEOSCLERL OUTFLOW:

Through this pathway, 10%-20% fluid is drained out into suprachoroidal space and drained by venous circulation of ciliary body and sclera.

Amount of secretion is 2.5 microlitres/min.

STEPS OF AQUEOUS FORMATION:

3 processes are involved namely,

1.Diffusion – it accounts for 10% of aqueous formation.

2.Ultra filtration- it accounts for 20% of aqueous formation.

3.Secretion- major part, 70% of aqueous is produced by this process.

1. Diffusion:

This process transports lipid soluble substances via lipid soluble membrane. This process is proportional to concentration gradient.

2. Ultrafiltration:

This process transports water soluble substances via micro pores in the cell membrane. This process is controlled by hydrostatic pressure, which is dependant on IOP,

Blood pressure in ciliary capillaries

Plasma oncotic pressure.

The above two mechanisms are passive processes.

3. Secretion:

As it is an active process, it requires energy. Large size water soluble molecules are transported by this process. It plays major part in aqueous secretion.

Total volume of aqueous fluid is 0.3 ml out of which

0.25ml is in anterior chamber

0.05 ml is in posterior chamber.

Refractive index 1.336.

The density of aqueous is slightly greater than water.

PH is 7.2. It is slightly hyperosmotic than plasma.

CONTENTS OF AQUEOUS:

- Water forms 98-99%
- Glucose – It is 70% of that in plasma. It nourishes cornea. Its concentration is increased in diabetics.
- Protein – it is lesser than that in plasma.
- Immunoglobulins- IgG is normally present in aqueous.

ORGANIC IONS:

- Ascorbate – It is 10 times higher than plasma, functions as antioxidant,
- Lactate.

INORGANIC IONS:

- Sodium, Potassium,
- Bicarbonate ions,
- Chloride ions,
- Calcium,
- Phosphate[2]

ANATOMY OF ANGLE STRUCTUTRES:

Angle recess is formed between posterior surface of cornea and anterior surface of iris bounded from anterior to posterior by

- Schwalbe's line,
- Trabecular meshwork,
- Scleral spur,
- Anterior surface of Ciliary body along with root of iris.

1.Schwalbe 's Line:

It is the posterior termination of descemet's membrane.

2.Trabecular Meshwork:

It consists of 3 parts,

uveal meshwork is the innermost,

corneoscleral meshwork is the intermediate layer,

juxtacanalicular tissue is the outer one adjacent to schlemm's canal. Juxta canalicular tissue offers maximum resistance to aqueous outflow.

3.Scleral Spur:

It is the thin lip of scleral projection which gives attachment to ciliary muscle. It accommodates schlemm's canal. Posteriorly it gives attachment to longitudinal ciliary muscle.

4.Ciliary Body Band:

- Is a greyish band,width depends on the level of iris insertion. It can be wider in myopes. It has 2 parts.
- Anterior pars plicata
- Posterior pars plana.

The outermost part is supra ciliary stroma and inner part is stroma which has ciliary muscle as the major component. The ciliary muscle fibres are Longitudinal fibres , Circular fibres and Radial fibres.

Nerve supply:

Ciliary ganglion supplies parasympathetic nerve supply to the Ciliarybody.

Vascular supply:

It is by major arterial circle. This circle is formed by the anastomosis of long and short posterior ciliary arteries

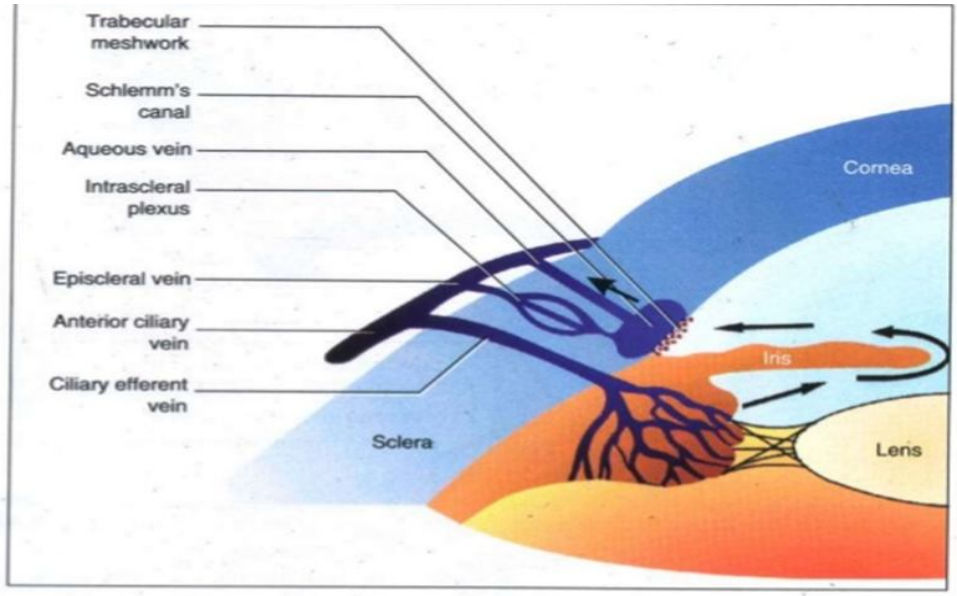

Figure 3 : Aqueous outflow pathway

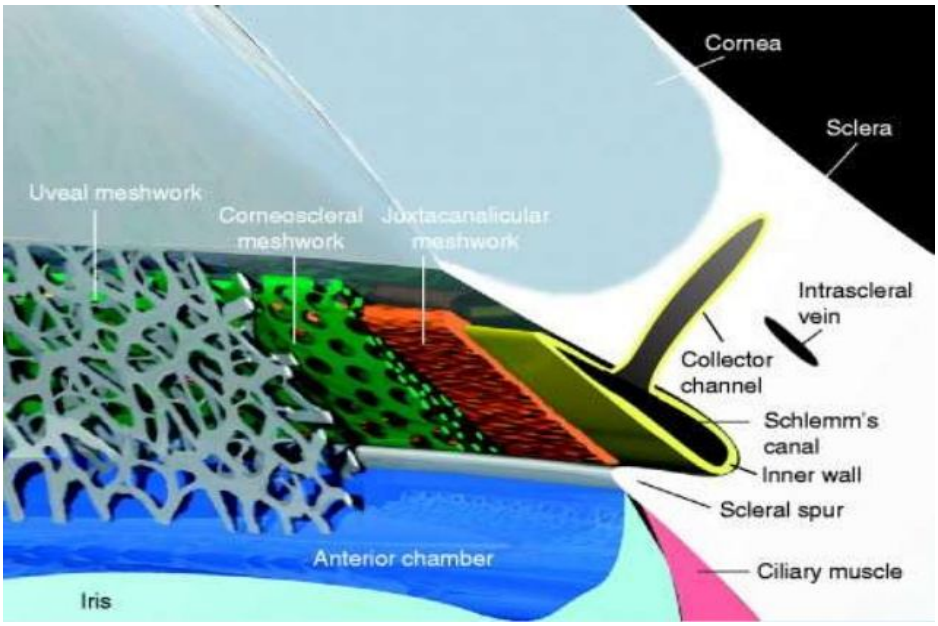

Figure 4 : Anatomy of trabecular meshwork

PIGMENTED AND NONPIGMENTED EPITHELIAL LAYER:

Pigmented layer is continued with retinal pigment epithelial layer. Nonpigmented layer is continued with pigment epithelial layer of iris.

SCHLEMM'S CANAL:

It is an endothelium lined channel.

COLLECTOR CHANNELS:

2 types are there.

- INDIRECT CHANNELS,
- DIRECT CHANNELS.

These channels drain into episcleral veins.

These episcleral veins drain into cavernous sinus via

Anterior ciliary veins and

Superior ophthalmic veins.

INTRAOCULAR PRESSURE:

Intraocular pressure is determined by aqueous production, resistance to aqueous outflow and episcleral venous pressure.

The circulation of aqueous maintains the Intraocular pressure. The distribution of intraocular pressure in our population is 11 to 21 mmhg. It can be measured by tonometry. Various types of tonometry are available to measure intraocular pressure. Intraocular pressure shows diurnal fluctuation.

Normally there is early morning peak and evening trough. Normal difference will be 6 mmhg. Patients with high diurnal fluctuation will have more progression of glaucoma. No sex difference found in the distribution of intraocular pressure[3].

FACTORS AFFECTING INTRAOCULAR PRESSURE:

1.AQUEOUS HUMOR PRODUCTION:

It is controlled by infection and inflammation of ciliary body, blood pressure and osmolarity of plasma. Beta blockers and carbonic anhydrase inhibitors have influence on intraocular pressure by affecting aqueous production.

2. AQUEOUS OUTFLOW RESISTANCE:

It is influenced by trabecular meshwork inflammation, clogging of trabecular mesh by inflammatory and cellular debris, exudates, pigments, tumor cells.Also pupillary block and peripheral anterior synechiae offers resistance to outflow. Outflow resistance is decreased by prostaglandins, miotics and adrenaline.

3. EPISCLERAL VENOUS PRESSURE:

Normal amount of episcleral venous pressure is 8 -10 mmhg. It is raised in sturge weber syndrome, where the glaucoma is refractory to routine medical management. It is raised also in thyroid orbitopathy, caroticocavernous fistula. Valsalva maneuver can also increase the episcleral venous pressure.

TONOMETRY:

It is the procedure used to measure intraocular pressure.

It can be

- Contact
- Noncontact tonometer.

In contact tonometer type, there are 2 varieties.

1. Indentation tonometer - schiotz,

2. Applanation tonometer which can be categorized into

a. VARIABLE FORCE AND CONSTANT AREA:

It comprises,

- Goldmann Applanation tonometer,
- Perkins,
- tonopen,
- pneumotonometer

b. CONSTANT FORCE AND VARIABLE AREA:

- Makalakov

NON-CONTACT TONOMETER:

- Pulse air

Figure 5 : Schiotz tonometer

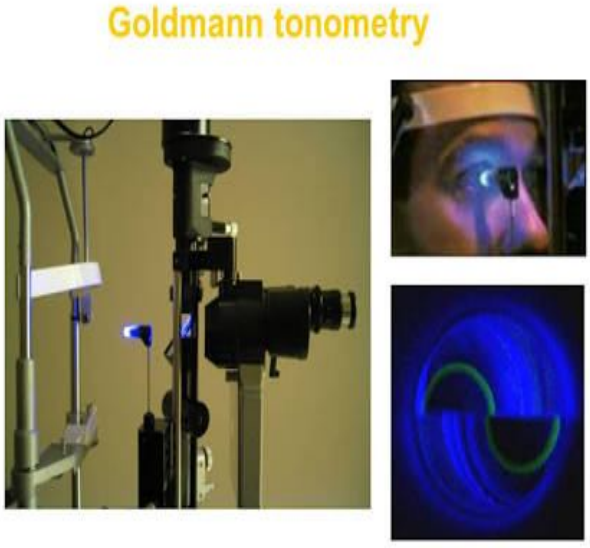

Figure 6 : Goldmann Applanation tonometry

NEWER ADVANCES:

Trans palpebral tonometer,

Dynamic contour tonometry.

Sensimed trigger fish.

Schiotz and Maklakov displace large amount of fluid,thereby raising the intraocular pressure significantly, so they are called high displacement type,these are less accurate than the low displacement one. But Goldmann and Mackay marg displace only very small amount of aqueous, so they are called low displacement tonometry type. Goldmann tonometer pushes only 0.5 microliter, so it increases only 3% increase in Intraocular pressure[2].

MECHANISM OF INDENTATION TONOMETER:

When the indentation tonometer is placed on cornea, additional forces are set up by outer coats of eye ball, so true intraocularpressure (p0) is further raised to P1.Therefore true IOP is derived from Fridenwald conversion table. The major demerit is false recordings were observed in patients with abnormal scleral rigidity

MECHANISM OF APPLANATION TONOMETRY

They measure intraocular pressure based on Imbert-Fick Law. Corneal rigidity and capillary attraction of tear meniscus forces influence IOP. If the area flattened is 3.06 mm^2, the above forces cancel them each other.

Among the applanation tonometers, Goldmann applanation is the gold standard tonometry. It contains biprism mounted on slitlamp measured with cobalt blue filter. Measurement is influenced by corneal thickness and astigmatism.

GONIOSCOPY:

The term GONIOSCOPY was termed by Trantas. It is the procedure to look for the angle status whether it is open or closed and to look for the presence of peripheral anterior synechiae, foreign body in the angle, blood in the angle.

Based on the angle status we can classify the glaucoma patients into

- Open angle glaucoma,
- Angle closure glaucoma,
- Occludable angles.

It has to be done for angle closure and occludable angles periodically.

Basic principle of this procedure is, the total internal reflection of light rays from the angle is eliminated with the help of gonioscope.

There are two types.

1.Direct Gonioscope

2.Indirect Gonioscope

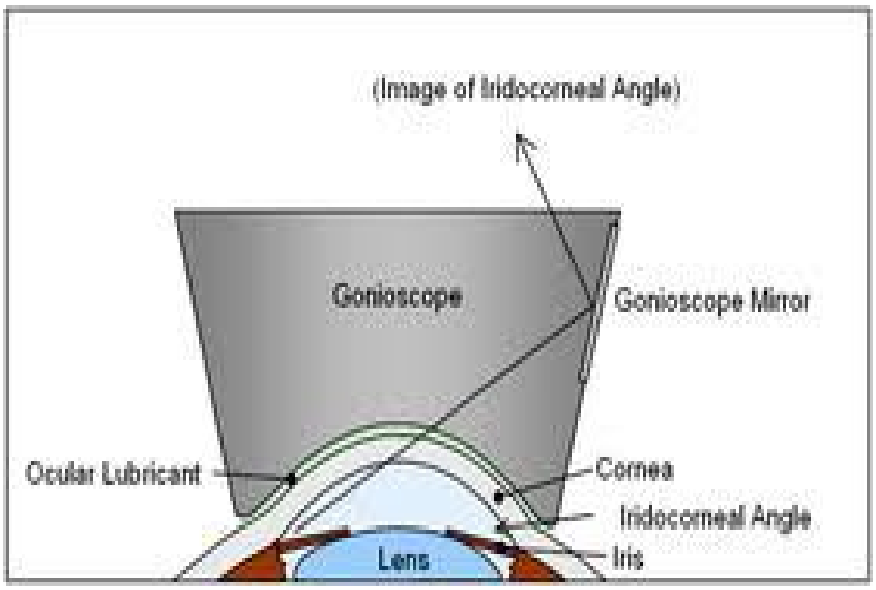

Figure 7 : Mechanism of gonioscopy

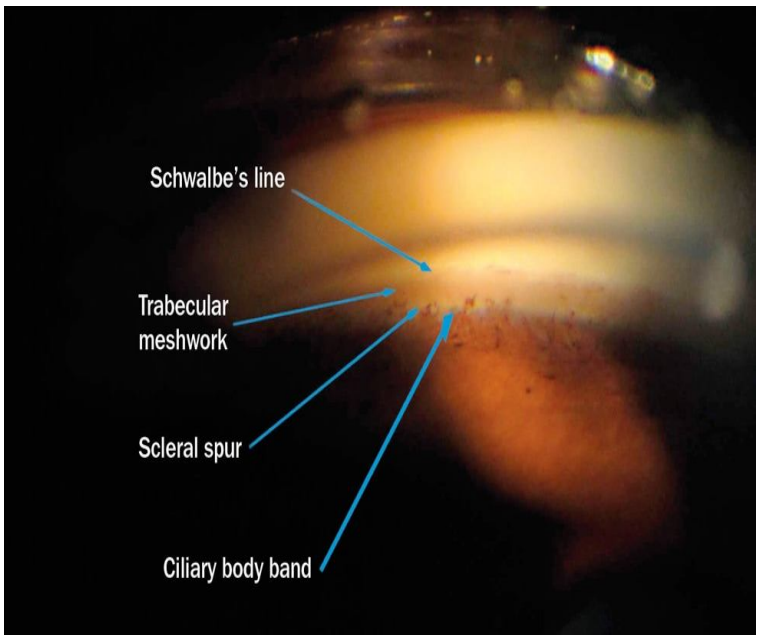

Figure 8 : Gonioscopic view of Angle Structures

1. DIRECT GONIOSCOPY:

- It is used with operating microscope.
- Koeppe lens – most commonly used lens, for diagnostic purpose.
- Swan jacob goniolens – also used for surgical purpose for children.
- Richardson shaffer's lens – small koeppe lens used for infants.
- Huskin's barkan lens – prototype of surgical goniolens.
- Worth goniolens - anchors to cornea with partial vaccum.

BENEFITS OF DIRECT GONIOSCOPY:

It provides a straight view in contrast to inverted view with indirect lens.

We can do a detailed examination of angle structures.

- By using two lenses, we can examine both eyes simultaneously.
- Panoramic view is obtained so that one part of angle can be compared with the other part.
- Flexibility is good.
- It can be used in goniotomy surgery.

DISADVANTAGES:

1) It is inconvenient to use and also time consuming.

2) Added benefits of slitlamp was not available.

Figure 9 : Koeppe Lens,
examination with koeppe lens

Figure 10: Swan Jacob lens,
examination with swan Jacob lens

INDIRECT GONIOSCOPY:

- Goldmann three mirror gonioprism:

  Mirror inclined at 59° - used for gonioscopy,

  67º – for examining pars plana of ciliary body,

  73º - to look for ora serrata.

- Goldmann two mirror gonioprism:

  Mirror inclined at 62º

- Goldmann single mirror prism:

  Mirror inclined at 64º

- Allen thorpe gonioprism:

  It has four prism instead of mirrors. we can see the whole angle without rotating the prism.

  Above lenses will require coupling agent.

GONIOLENS NOT REQUIRING COUPLING AGENT:

- Zeiss four mirror – Mirrors inclined at 64º, indentation gonioscopy can be done.
- Posner - It has detachable handle
- Sussman- It has no handle.

Figure 11 : Goldmann single and three mirrorgoniolens

Figure 12 : Sussman four mirror lens

Figure 13 : Zeiss four mirror lens

FUNDUS CHANGES IN GLAUCOMA:

Normal optic disc is vertically oval, 1.5 mm in diameter. Neuro retinalrim lies between disc margin and cup. Normally it will be pink in color. It follows ISNT rule. That implies that inferior neuro retinal rim is the broadest. The next one is superior, nasal and temporal neuro retinal rim. As glaucoma advances, neuro retinal rim will be thinned out and lost. Cup represents absence of axons. Pallor of cup indicates loss of glial support.

CUP DISC RATIO:

It is expressed as diameter of the cup as a fraction of diameter of optic disc. vertical cup disc ratio is more important than horizontal cup disc ratio,because pores are larger in superior and inferior poles and also less glial support to superior and inferior pole areas.

Cup disc ratio of > 0.3 or Asymmetry of cup disc ratio >0.2 between two eyes are considered significant.

Disc size is also important while measuring cup disc ratio.

OPTIC NERVE HEAD CHANGES IN GLAUCOMA:

NOTCHING:

Focal loss of neuroretinal rim results in **notching**, most common in inferotemporal quadrant,which is more specific of glaucoma

BAYONETTING:

It means double angulation of a blood vessel. As neuroretinal rim thins out, blood vessel crossing the rim will double angulate resulting in bayonetting.

LAMINOR DOT SIGN:

It occurs due to enlargement of fenestrae in lamina cribrosa.

It appears as greyish dots.

TEMPORAL UNFOLDING:

When loss of neuroretinal rim starts temporally, proceeds circumferentially, it is called as temporal unfolding with concentric atrophy.

BEAN POT CUPPING:

Advanced cupping with total loss of neuroretinal rim results in bean pot cupping.

SPLINTER HEMORRHAGES:

Hemorrhage which crosses disc margin mostly seen in inferotemporal quadrant It signifies the progression of the disease, most commonly seen in normotensive glaucoma. It is also called Drance hemorrhages.

BARRING OF CIRCUMLINEAR VESSEL:

As the neuroretinal rim recedes, normal circumlinear vessel which outlines the cup will be barred from cup margin.

VERTICAL ECCENTRICITY OF CENTRAL RETINAL VESSEL TRUNK

RETINAL NERVE FIBRE LAYER

Retinal nerve fibre layer will be seen as striations in light reflex. On red free filter, retinal nerve fibre layer defects can be visualized as darker areas of slit or wedgeshaped defects.

PERIPAPILLARY PIGMENTATION:

There are 2 zones, alpha and beta zones.

Beta zone lies between outer alpha and optic disc margin. It occurs due to retraction of retinal pigment epithelial layer due to atrophy and choroidal degeneration. Zone beta corresponds with visual field defects.

Zone alpha is the outer zone,which lies external to beta zone. It occurs due to retinal pigment epithelial hyper or hypo pigmentation[6].

OPTIC DISC EVALUATION:

It can be done with direct ophthalmoscopy, indirect ophthalmoscopy, slit lamp biomicroscopy with 90 d or 78 d lens, fundus camera photography. It can be measured quantitatively by confocal scanning laser tomography, also known as Hiedelberg retinal tomography, scanning laser polarimetry, optical coherence tomography. With every follow up, following findings should be noted.

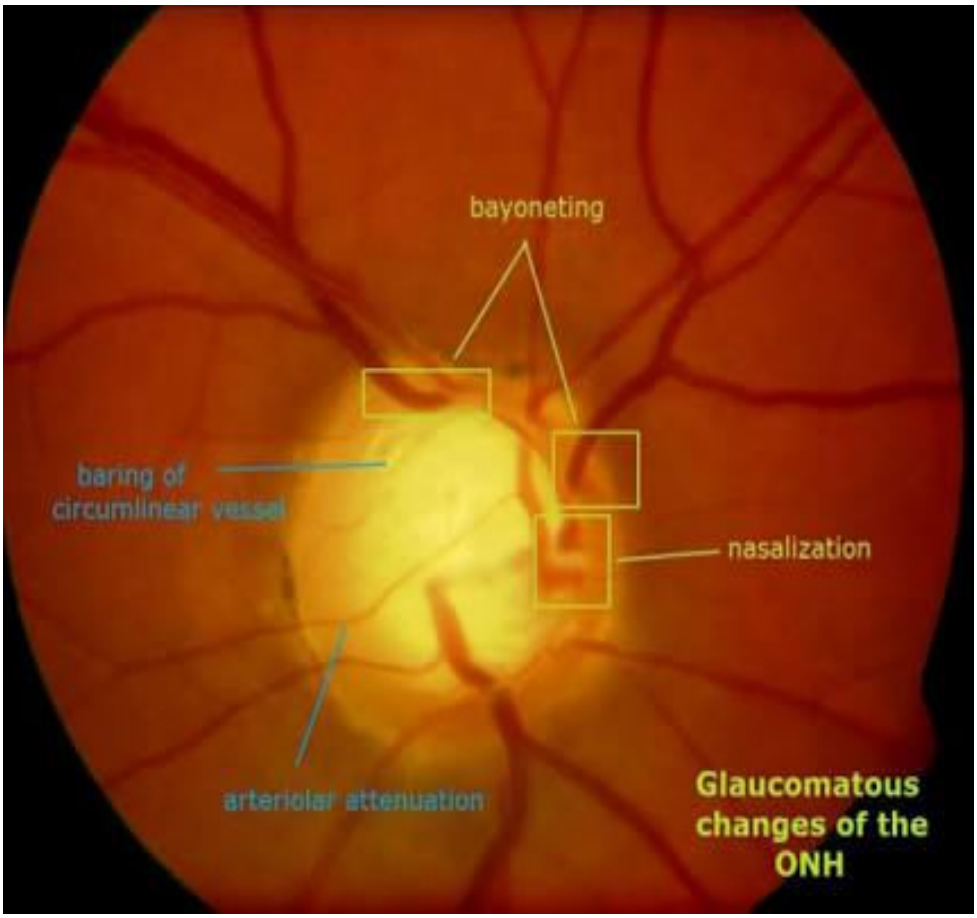

Figure 14 : Glaucomatous changes of optic disc

1. Optic disc size and shape
2. Optic cup size and shape
3. Neuroretinal rim
4. Disc hemorrhages
5. Blood vessel changes
6. Peripapillary changes
7. Retinal nerve fibre layer changes.

ASSESSMENT OF GLAUCOMA PATIENT:

Glaucoma assessment starts with History taking. Proper history taking is very important in diagnosing glaucoma early. Any family history of glaucoma in the family members should arouse suspicion. Any history of frequent change of presbyopic glasses, colored halos around light, headache, delayed dark adaptation and any chronic usage of topical drops, topical medications needs further investigation.

Screening of the population includes,

- IOP examination,
- Fundus examination,
- Visual field testing by supra threshold strategies.

CLASSIFICATION OF GLAUCOMA:

It has been classified as

- primary congenital glaucoma which is present since birth and there are not accompanied with any systemic and ocular disease.
- Developmental glaucoma is termed when the glaucoma is associated with ocular and systemic diseases.
- Juvenile glaucoma is termed when glaucoma occurs after 3 years of age.

Primary congenital glaucoma occurs due to involvement of CYP1B1 gene. Juvenile glaucoma is associated with mutation of TIGR.

Primary congenital glaucoma is characterized by blepharospasm, watering,pain. On examination corneal diameter is increased with corneal edema, haab's striae due to breaks in decemat's membrane.

Gonioscopy shows anterior insertion of iris. IOP is characteristically elevated. Due to stretching and enlargement, axial length will be increased resulting in axial myopia, zonular dialysis, eventually subluxation of lens. Due to axial myopia, the child will go for amblyopia. Decemat's membrane break can cause astigmatism. Patient is managed initially with IOP lowering drugs. But surgery is the mainstay of treatment. Goniotomy can be done if the cornea is clear. Trabeculotomy is planned if there is corneal edema.Trabeculotomy can be combined with trabeculectomy.

SYNDROMES AND GLAUCOMA:

- Sturge weber syndrome,
- Microspherophakia,
- Pierre robinson syndrome,
- Lowe's syndrome,
- Homocystinuria,
- Marfan's syndrome,
- Neurofibromatosis,
- Aniridia,
- Peters anomaly[2].

Primary glaucoma can be either primary open angle glaucoma and primary angle closure glaucoma.

Open angle glaucoma could be primary open glaucoma, normal tensive glaucoma, and ocular hypertension.

Secondary open angle glaucoma can be classified as,

- Pseudo exfoliation glaucoma,
- Pigmentary glaucoma,
- Uveitic glaucoma,
- Intra ocular tumors causing glaucoma,
- Intra ocular hemorrhage causing glaucoma,
- Lens induced glaucoma
- Traumatic glaucoma,
- Steroid induced glaucoma,
- Retinal detachment associated glaucoma.

Secondary angle closure glaucoma could be classified as

- Anterior pulling mechanism due to,

  ICE syndrome,

  Fibrous ingrowth, epithelial down growth.
- Posterior pushing mechanism due to,

  Ciliary body cysts and tumors,

  Choroidal effusion and detachment,
- Secondary glaucoma with pupillary bock,
- Aqueous misdirection syndrome
- Lens induced glaucoma.

PATHOGENESIS OF GLAUCOMA:

Pathogenesis of glaucoma could be either due to mechanical or vascular etiology.

MECHANICAL DAMAGE:

It is due to chronic increased IOP causing direct damage, or impaired delivery of nutrients or neurogenic factors.

VASCULAR DAMAGE:

It can be due to impairment of auto regulation. Vascular factors like Nitric oxide and endothelin also play role. NTG is more common in patients with ischemic and vasospastic disease.

There can be genetically determined apoptosis- programmed cell death.

Glutamate induced NMDA receptor activation.

SYSTEMIC FACTORS ASSOCIATED WITH GLAUCOMA:

- Diabetes
- Hypertension,
- Migraine,
- Grave's disease
- Cushing's syndrome.

OCULAR FACTORS ASSOCIATED WITH GLAUCOMA:

- Myopia,
- CRVO
- central corneal thickness[2].

SYMPTOMS AND SIGNS OF GLAUCOMA:

In the early stage, there won't be any symptoms.

Gradually there can be

- Mild pain,
- Headache,
- Colored halos around the light,
- Delayed dark adaptation,
- Peripheral constriction of fields.

SIGNS OF GLAUCOMA:

In POAG, slit lamp examination of anterior segment will be normal. Gonioscopy will show open angles.

IOP will be elevated.

VISUAL FIELD DEFECTS IN GLAUCOMA:

Standard automated perimetry is done by Humphery field analyzer or Octopus.

Programs used are,

- 30-2,
- 24-2,
- 10-2,
- Macular program,
- Nasal step program.

In 30-2, total points tested are 76 points in 30degree field, 6degrees apart.

In 24-2, total points tested are 54 in 24degree field, 6degrees apart.

In 10-2 total points tested are 68 points in 10degree field, 2degrees apart.

In Macular program, 16 points are tested in central 5degree, 2 degrees apart.

FIXATION TARGET:

- Central small diamond.
- Large diamond and
- Bottom LED are used for patients with central scotoma.

TARGET SIZE:

Goldman size 3 and 5

TESTING STRATERGY:

OCTOPUS:

3 strategies are,

- Normal
- Dynamic
- TOP- It is tendency oriented perimetry.

HUMPHERY FIELD ANALYSER:

It has three strategies

- SITA- it is swedish interactive threshold algorithm.
- SITA FAST
- FULL THRESHHOLD.

RELIABILITY FACTORS:

- Fixation loss should be less than 20%,
- False positive should be less than 33%.
- Similarly false negative should be less than 33%

Visual field printout will be showing

- Patient data and refraction
- Test parameters and test programs, strategies
- Grey scale
- Value table
- Corrected data and corrected comparison data
- Probability plot and corrected probability plot
- Bebis curve
- Global indices

In Octopus, it includes

- Mean sensitivity,
- Mean defect,
- Loss variance,
- Short term fluctuation,
- Corrected loss variance.

In Humphery,

- Glaucoma hemifield test,
- Mean deviation,
- Patterned standard deviation,
- Corrected patterned standard deviation,

- Short term fluctuation.

FIELD DEFECTS:

- Isolated para central scotoma is the early defect.
- It can evolve into arcuate scotoma,
- Bi arcuate scotoma.
- Ring scotoma
- Nasal step defect.
- Advanced field defects sparing temporal island of vision can be seen in end stage.

PREPERIMETRIC GLAUCOMA:

Normally visual field defects appear when there is 20-40% loss of retinal ganglion cell death occurs. So early detection of retinal nerve fibre layer loss diagnoses glaucoma at early stage[12].

RECENT ADVANCES IN PERIMETRY:

- Short wavelength automated perimetry-

  It is also known as SWAP, Blue on yellow perimetry. By this method, we can detect glaucoma early than standard automated perimetry.

- High pass resolution perimetry:

  It is also known as ring Perimetry, it uses rings instead of spots.

- Flicker perimetry:

The stimulus in this perimetry is flickering stimulus instead of static flashes used in other types.

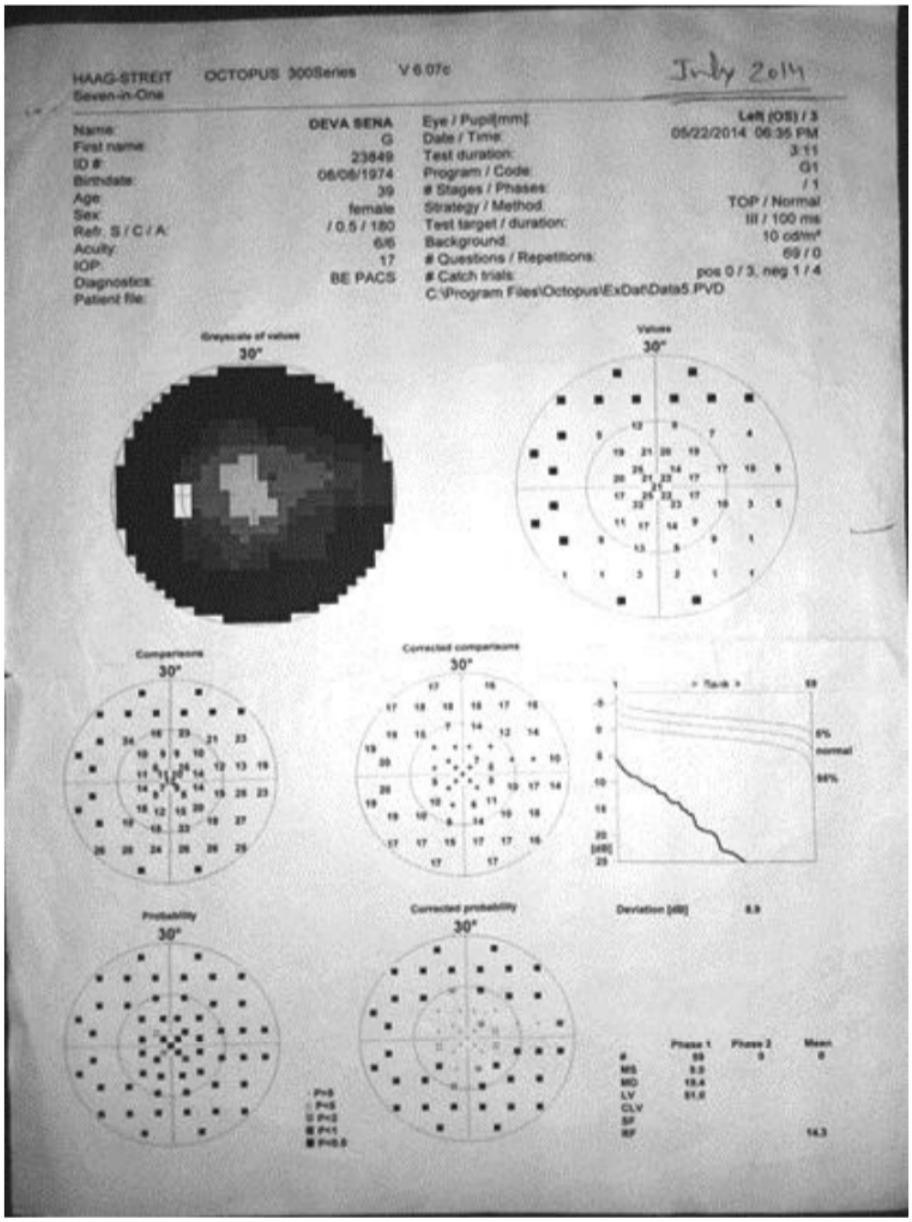

Figure 15 : Octopus perimetry of a patient showing bi-arcuate field defects

MANAGEMENT:

It includes

- Medical management,
- Laser treatment,
- Surgical management.

MEDICAL MANAGEMENT:

It acts by

- Suppressing aqueous production,
- Increasing outflow,
- Neuro protective drugs.

CLASSIFICATION OF ANTIGLAUCOMA DRUGS:

It is classified as

- B – blockers,
- Adrenergic agonists,
- Prostaglandin analogue,
- Carbonic anhydrase inhibitors
- Hyper osmotic agents,
- Miotics.

SYMPATHOMIMETIC DRUGS:

It includes,

1.EPINEPHRINE:

It acts on both alpha and beta receptors.

2.DIPIVEFRINE:

It is a prodrug, which is converted to epinephrine which acts on alpha and beta receptors.

3.CLONIDINE:

It is a centrally acting antihypertensive agent. In glaucoma it acts by decreasing aqueous humor production by acting on alpha receptors.

4.BRIMONIDINE (0.2%)

It is a selective alpha 2 adrenergic agonist. This drug has triple action.

- It decreases aqueous production,
- It increases aqueous outflow also,
- It has neuro protective action.

5.APRACLONIDINE:

It is used after laser procedures to prevent post IOP spike following,

- Laser trabeculoplasty,
- YAG laser iridotomy,
- YAG capsulotomy.

BETA ADRENERGIC BLOCKERS:

It includes,

1.TIMOLOL:

It is the most commonly used drug. First choice drug in POAG unless contraindicated. It is the nonselective beta blocker. It decreases aqueous secretion by blocking beta 2 receptors in ciliary body.

2.LEVOBUNOLOL:

It is also a nonselective beta blocker similar to timolol.

3.BETAXOLOL:

It is a cardio selective beta 1 blocker, thereby safely used in Bronchial asthma patients. It is most commonly used in congenital glaucoma.

4.CARTEOLOL

5.METIPRANOLOL

Beta blockers are contraindicated in bradycardia, heart failure, arrythmias, Respiratory diseases like bronchospasm, obstructive airway disease.

CARBONIC ANHYDRASSE INHIBITORS:

It includes,

1. ACETAZOLAMIDE
2. METHAZOLAMIDE,
3. DORZOLAMIDE,
4. BRINZOLAMIDE.

These drugs inhibit carbonic anhydrase enzyme which is essential for aqueous humor production. But these drugs can cause electrolyte imbalance, gastric ulcer, urinary disturbance. Acetazolamide is available as tablets. It is available in 250 mg strength.

HYPEROSMOIC AGENTS:

1.GLYCEROL:

This oral hyperosmotic agent is available in 50% solution. Dosage can be 1 gm/kg body weight. Usually it is given as a mixture with lemon juice or water. But it is contraindicated in diabetic patients with glaucoma.

2.MANNITOL:

It is an intravenous drug, used in very high intraocular pressure and acute angle closure attack. Dosage is 1gm /kg body weight. It is available in 20% solution. It can be given safely in diabetic patients. But caution required in patients with cardiac and pulmonary edema patients, as it can precipitate pulmonary edema.

PROSTAGLANDIN ANALOGUES:

1. LATANOPROST:

It is available in 0.005%. It acts by increasing uveoscleral outlet, thereby reducing intraocular pressure.

2. BIMATOPROST:

It is available in 0.03%

3. TRAVOPROST:

It is available in 0.004%

4. UNOPROSTONE:

It is available in 0.12%. It is PG F2 alpha.

Prostaglandin analogues increases uveoscleral outflow. Because of long duration of action, it can be given as once daily dosage. But it causes conjunctival hyperemia, periocular pigmentation, hypertrichosis, iris hyperpigmentation. It is also known as prostaglandin related periorbitopathy[13].

LASER TREATMENT:

Laser procedures are,

Laser Trabeculoplasty,

Laser Iridotomy,

Laser Iridoplasty,

Cyclophotocoagulation.

1. LASER TRABECULOPLASTY:

It can be

- Argon Laser Trabeculoplasty
- Selective Laser Trabeculoplasty
- Micropulse Laser Trabeculoplasty.

INDICATIONS

- Pseudo exfoliation glaucoma,
- Pigmentary glaucoma,
- Chronic open angle glaucoma.

ARGON LASER TRABECULOPLASTY:

- It uses power of 300- 1200 mW,
- spot size is 50 µm,
- 180 degrees of trabecular meshwork can be treated simultaneously.
- 30-50 spots applied.

End point is transient blanching to bubble formation.

Complications of Argon Laser Trabeculoplasty are scarring of trabecular area, eventually resulting in failure of trabeculoplasty.

SELECTIVE LASER TRABECULOPLASTY:

In this procedure, frequency doubled ND: YAG laser is used. It selectively targets pigmented trabecular meshwork, without damaging nonpigmented trabecular meshwork.

- The spot size is 400 microns
- Frequency of laser is 532 nm,
- Power is 0.8 mJ
- Duration is 3 nano sec,
- 50 spots are applied,
- 180-360 degree can be treated simultaneously.

The main advantage over argon laser trabeculoplasty is, it is a repeatable procedure. There is no scarring.

MICROPULSE LASER TRABCULOPLASTY:

This technology breaks the continuous wave into short pulses, that allow cooling. So it reduces thermal damage.

- It uses 532 nm wavelength
- power is 1000 mW,
- spot size 300 µm,
- 15% duty cycle,
- 360º can be treated simultaneously.

COMPLICATIONS:

It includes

- Transient IOP spikes,
- Iritis,
- Inadvertent damage to cornea, lens, fovea.
- Hemorrhage.

But laser trabeculoplasty is contraindicated in angle closure and uveitic glaucoma.

LASER IRIDOTOMY:

- It is indicated in angle closure suspects, angle closure glaucoma, creeping angle closure glaucoma.
- Laser used is ND: YAG,
- Lens used are Abraham lens and Wise lens.
- Placement of laser- superior iris 11-1 o'clock.

- Patient should be instructed to apply pilocarpine drops to stretch iris.

LASER IRIDOPLASTY:

It is most commonly done in pleateau iris syndrome.

OTHER LASER PROCEDURES:

- Co2 Laser assisted Sclerectomy Surgery,
- Laser Suturelysis
- Laser Synechiolysis,
- Gonio Photocoagulation
- Goniopuncture.

SURGICAL PROCEDURES:

The filtration procedures includes,

- External filtration surgeries,
- Internal filtration surgeries.

EXTERNAL FILTERING SURGERY:

It can be further classified as,

- Full thickness procedures,
- Partial thickness procedures.

Trabeculectomy was first introduced by Cairns in1968. It creates fistula at limbus, so that there is a direct communication between anterior chamber and subconjunctival space. It bypasses trabecular meshwork and schlemm's canal.

INDICATION:

- Rapid progression of field defects,
- Medically uncontrolled glaucoma,
- Poor compliance with medical therapy,
- Intolerance to medical therapy due to side effects of antiglaucoma drugs[11].

STEPS OF TRABECULECTOMY PROCEDURE:

ANASTHESIA:

It is by retrobulbar, peribulbar, subtenon, subconjunctival anasthesia.

In paediatric cases, general anasthesia is preferred.

SUPERIOR RECTUS BRIDLE SUTURE,

CONJUNCTIVAL FLAP:

It has 2 types

- Limbal based conjunctival flap,
- Fornix based flap.

CHARACTERISTICS OF LIMBAL BASSED FLAP:

- Difficult to perform,
- Exposure is not good
- Difficult to make releasable sutures,
- Conjunctival handling is comparatively high,
- Antimetabolite application is relatively easy,
- Bleb appearance is ring of steel.

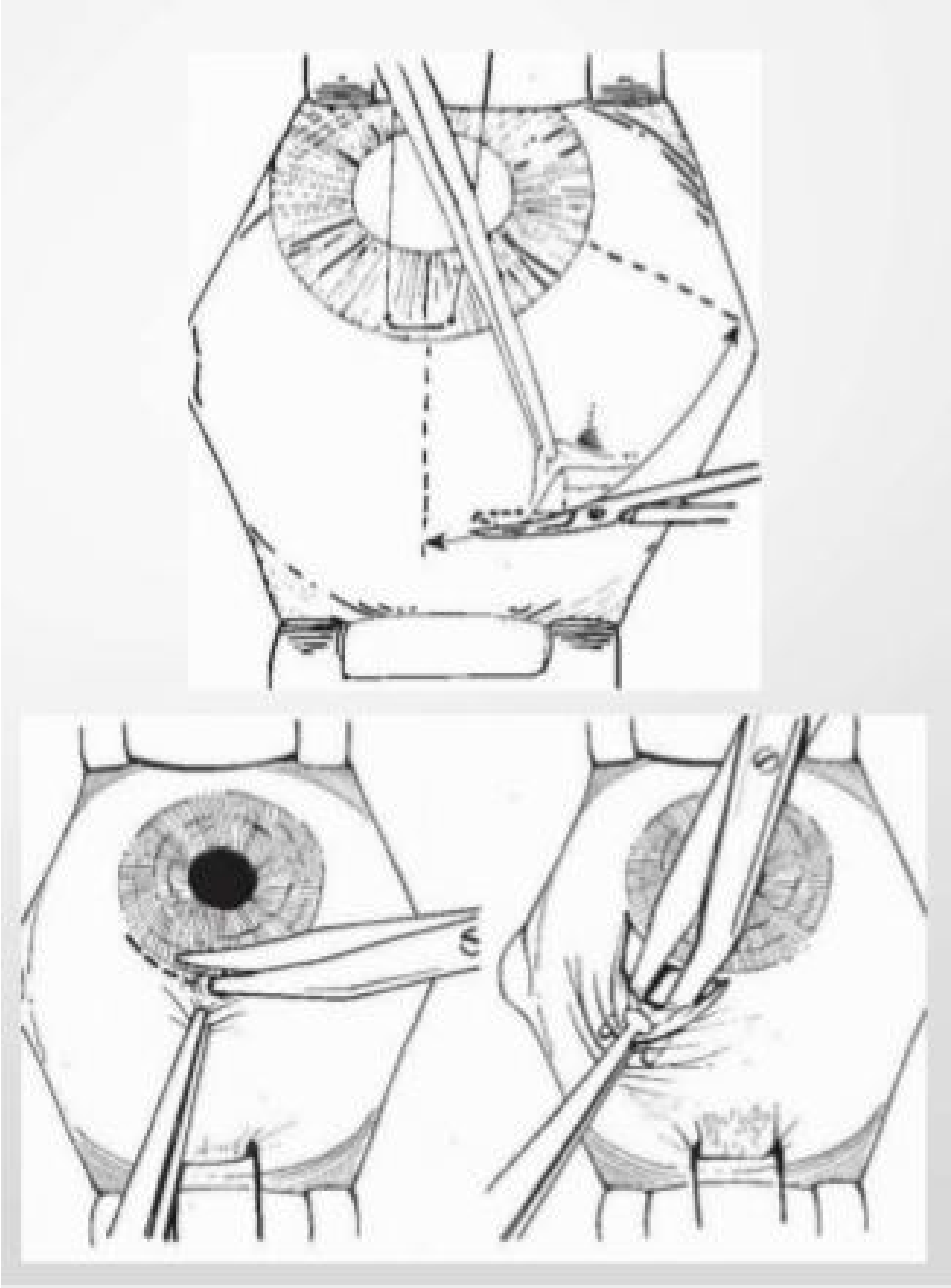

Figure 16: Conjunctival flaps

CHARACTERISTICS OF FORNIX BASSED FLAP:

- This flap creation can be easily performed,
- Exposure will be adequate.
- Antimetabolites application needs great caution as it causes necrosis of adjacent tissues.
- But conjunctival handling will be less, scarring is less, possibility of re operation is there.
- Bleb appearance will be posteriorly draining diffuse bleb.

SCLERAL FLAP DISSECTION:

- Partial thickness scleral flap, either triangular or rectangular in shape.

CONTROLLED PARACENTESIS:

- It is the very essential step, as it prevents sudden drop in IOP, thereby preventing hypotonic maculopathy,
- Also it is used for AC formation,
- continuous IOP maintenance by irrigation with saline or viscoelastic substance[2].

SCLEROSTOMY:

- It is done with kelly's punch or manually removed.

PERIPHERAL IRIDECTOMY;

- It should be typically a wider PI, extending beyond sclerostomy margins.
- So it prevents incarceration of iris pillars into sclerostomy site.

CLOSURE OF WOUND:

- Scleral flap suturing with 10-0 nylon sutures. It can be either
- Fixed sutures,
- Adjustable sutures,
- Releasable sutures.

LASER SUTURE LYSIS:

It is done to enhance filtration, done within 3 weeks,

- If anti metabolites are used, it can be done within 8 weeks.
- It is done with the help of
- Hoskins lens,
- blumenthal lens,
- volk lens,
- Done with argon laser

COMPLICATIONS:

INTRAOPERATIVE:

- Buttonholing of conjunctiva,
- scleral flap damage,
- Hemorrhage,
- supra choroidal hemorrhage.

POST OPERATIVE COMPLICATIONS:

- HYPOTONY WITH FLAT AC;

It occurs due to,

Bleb leak,

Over filtration and

Choroidal effusion.

- HYPOTONY WITH DEEP AC:

  It is due to over filtration.

- ELEVATED IOP WITH FLAT AC:

  It is due to,

  Aqueous misdirection,

  Pupillary block,

  Delayed suprachoroidal hemorrhage

- ELEVATED IOP WITH DEEP AC:

  It is due to bleb failure by,

  Internal blockade of ostium,

  Encapsulated bleb[4].

AIM & OBJECTIVES

Aim

To clinically analyse the Association of systemic factors in primary open angle glaucoma.

Objectives

- Incidence of association of systemic factors (diabetes, hypertension, dyslipidemia, thyroid disorder, anemia, ischemic heart disease and others)in primary open angle glaucoma.
- Comparing the progression of Primary Open Angle Glaucoma in Patients with systemic factors and without systemic factors

Subject Selection

100 patients of primary open angle glaucoma in the age group of 40-70 years attending glaucoma services at regional institute of ophthalmology were included in the study.

Inclusion criteria

- Age group 30-70 years
- IOP>21 mm of Hg
- Angles more than 270° should be > 3 (schaffers grading) in both eyes
- Patients having Glaucomatous field defect on automated perimetry

Exclusion criteria

- Age <30 years and >70 years
- Patients with angle closure

- Patients with secondary glaucoma

Analysis plan

- Demographic pattern of glaucoma in patients with systemic diseases
- Progression of glaucoma
- To analyse course of primary open angle glaucoma
- To analyse the etiology of primary open angle glaucoma
- To analyse the clinical profile of primary open angle glaucoma
- Treatment modalities
- Outcomes following treatment

Methodology

All were subjected to detailed anterior segment examination, best corrected visual acuity, intraocular pressure measurement either by Goldmann applanation tonometry or in cases with presence of corneal edema rebound tonometry reading was recorded, fundus examination, gonioscopic examination and visual field analysis will be done. Surgical procedures like trabeculectomy, antiglaucoma device and laser trabeculoplasty were done where necessary

Follow up

All patients are monitored regularly every three months to look for progression of glaucoma. At each visit visual acuity measurement, anterior segment examination by slit lamp, intraocular pressure by applanation tonometer, fundus examination and visual field analysis will be done.

Screening procedures

- Detailed history of present illness, associated systemic illness
- Complete general examination of the patient and vitals measurement
- Visual acuity using Snellen's acuity chart
- Complete examination of anterior segment and posterior segment
- Intraocular pressure measurement
- Direct and Indirect ophthalmoscopy
- Fields
- Random blood sugar, Blood Pressure
- Lipid profile
- Thyroid profile
- Complete hemogram
- ECG, Echo cardiogram

OBSERVATION AND ANALYSIS:

200 eyes of 100 patients with intraocular pressure more than 21 mmhg on all occasions were taken for the study.

AGE DISTRIBUTION:

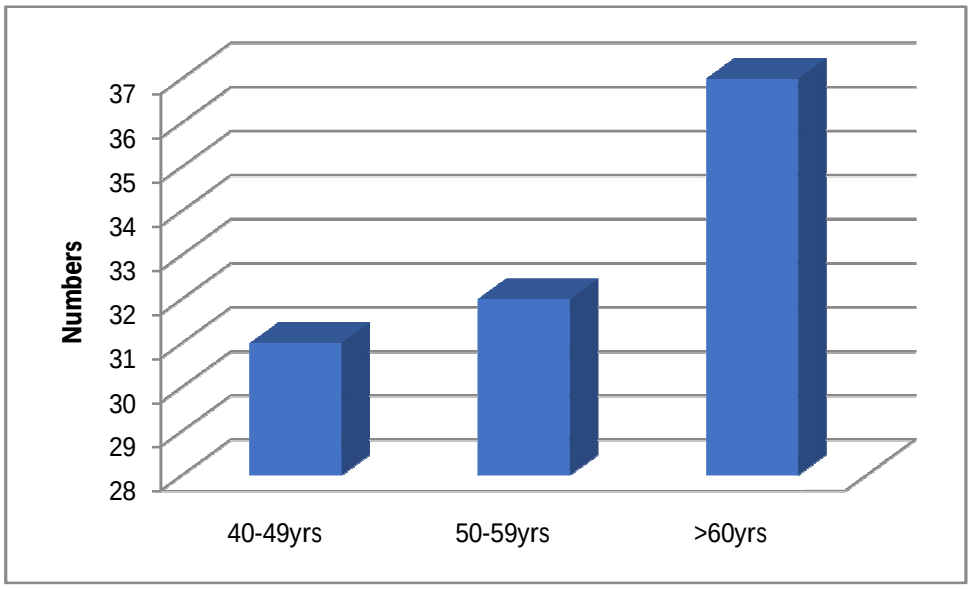

Chart 1 showing Age distribution

The ages of patients in our study varied from 40-70 years. Among 100 patients, 31 patients were between 40-49 years, 32 patients were in the age group of 50-59 years, 37 patients were in the age group of > 60 years. So the average age incidence is around 50-60 years. The possible reason for this age incidence is that most of this age group patients visit to hospital for refractive error or cataractous changes. Primary open angle glaucoma is detected by intraocular pressure measurement and fundus examination as part of routine examination.

SEX DISTRIBUTION:

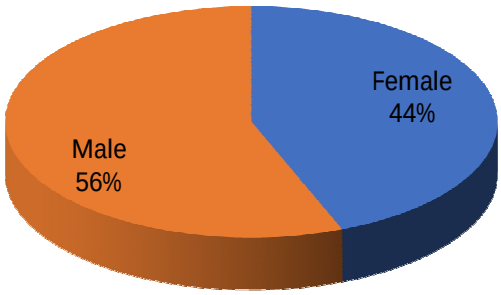

Chart 2 showing Sex distribution

In this study, 56% were males and 44% were females.

This could be due to high awareness regarding eye diseases and their need for refraction correction for their daily work purpose. Hence in our country, detection of glaucoma in males is more possible.

VISUAL ACUITY DISTRIBUTION:

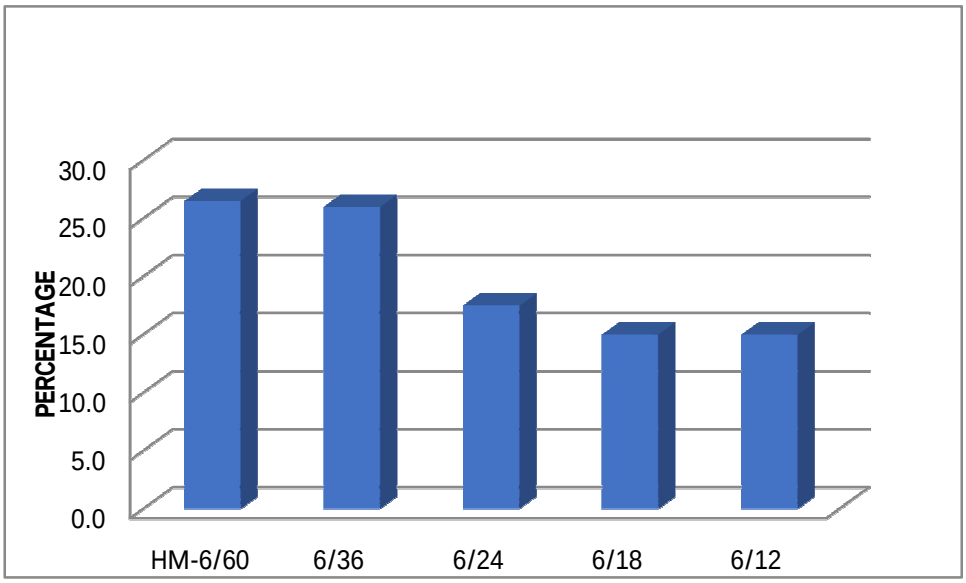

Chart 3 showing Visual acuity distribution

147 eyes (73.5%) were presented with visual acuity of 6/36 or better. 52 eyes (26%)were having 6/36, 35eyes (17.5%) were having 6/24, 30eyes (15%) were having 6/18, 30eyes (15%) were having 6/12.

53 eyes (26.5%) were presented with visual acuity of 6/60 or worse. The defective vision was due to refractive error in 12 eyes (22%), cataractous changes in 23eyes (44%), posterior capsular opacity in 2 eyes (4%) and due to glaucomatous optic atrophy in 16 eyes (30%). The defective visual acuity could be the reason for the need of these patients to visit hospital.

INTRAOCULAR PRESSURE DISTRIBUTION:

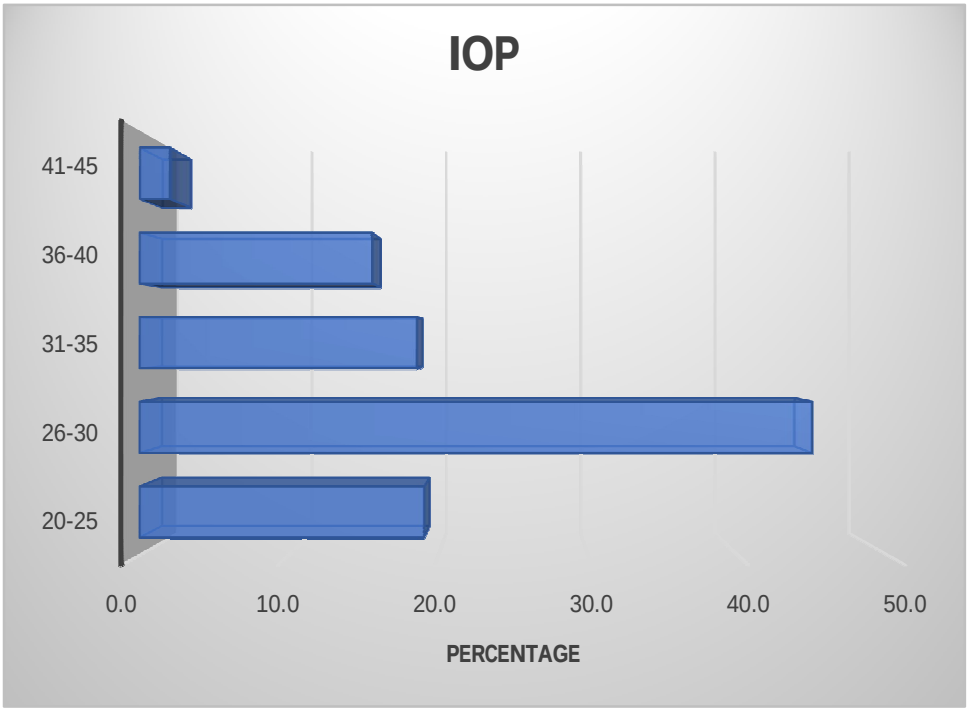

Chart 4 showing IOP distribution

In this study, out of 200 eyes, 38eyes (19%) presented with IOP in the range of 20-25 mmhg. 90eyes (45%) were in the range of 25-30 mmhg. 37eyes (18.5%) were in the range of 31-35 mmhg. 31 eyes (15.5%) were had 36-40 mmhg. 4 eyes (2%) had >40 mmhg.

So the mean intraocular pressure was 30mmhg.

FUNDUS CHANGES:

CUP DISC RATIO DISTRIBUTION:

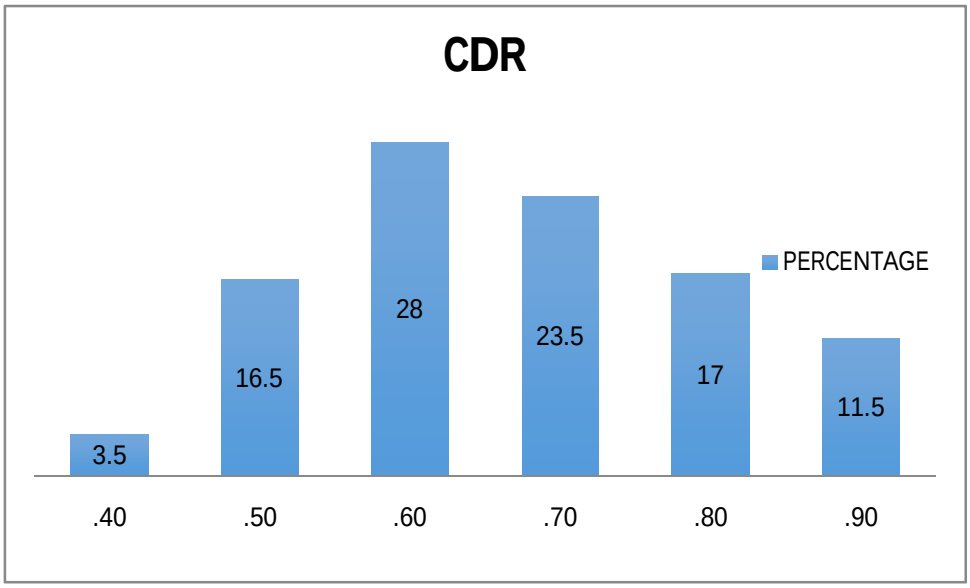

Chart 5 showing CDR distribution

In this study, on fundus examination,

23 eyes (11.5%) had CDR of 0.9,

34 eyes (17%) had cup disc ratio of 0.8,

47 eyes (23.5%) had CDR of 0.7,

56eyes(28%) had CDR OF 0.6,

33eyes (16.5%) had CDR of 0.5.

7eyes (3.5%) had CDR of 0.4.

In this study, 57 eyes (28.5%) were having advanced cupping (0.8 – 0.9)

FUNDUS

FUNDUS CHANGES	PERCENTAGE OF EYES	NO OF EYES
NASALISATION OF VESSELS	62.00%	124
BAYONETTING OF VESSELS	56.00%	112
LAMINOR DOT SIGN	66.00%	132
BARRING OF CIRCUMLINEAR VESSELS	22.00%	44
FOCAL NOTCHING	58.00%	116
PARAPAPILLARY ATROPHY	28.00%	56
RNFL DEFECTS	3.00%	6

Table 1 showing Fundus findings

VISUAL FIELD DEFECTS DISTRIBUTION:

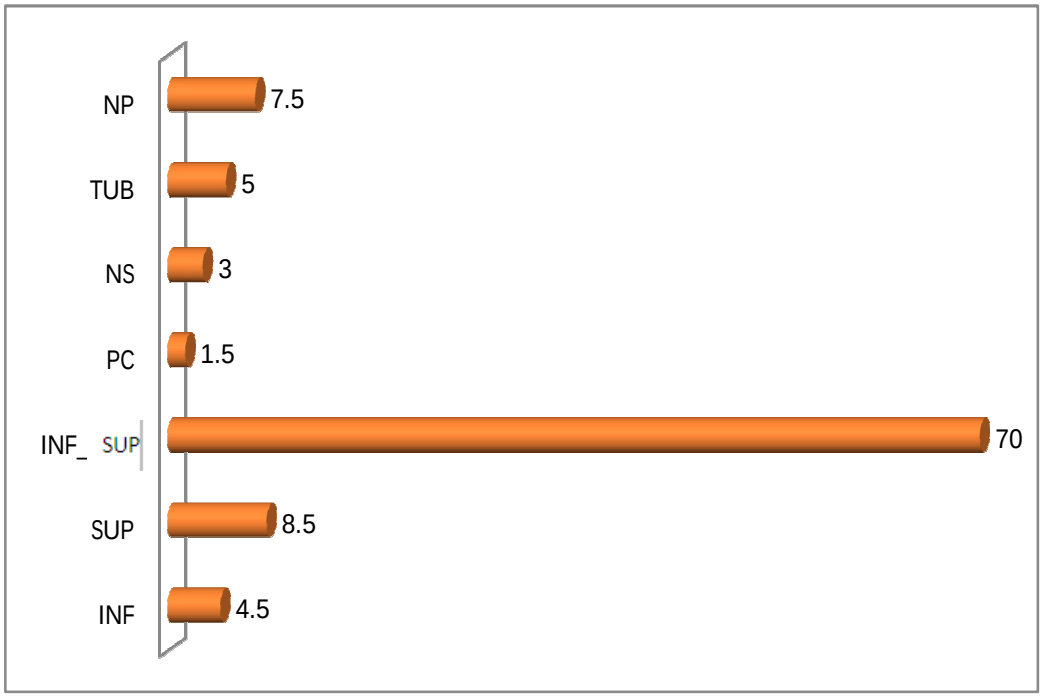

Chart 6 showing Field defects distribution

In our study, 140 eyes (70%) were presented with visual field defects in both superior and inferior arcuate regions. This is because of the fact that most patients were presented in the advanced stage of the disease. 6 eyes (3%) had nasal step, 3 eyes (1.5%) had paracentral field defects, 10 eyes (5%) had tubular field. Fields were not possible in 14 eyes (7.5%) due to poor vision.

- The absolute field defects were more than relative field defects. This also indicates the advanced stage of presentation.

DISTRIBUTION OF HYPERTENSION:

CATEGORY	SYSTOLIC (mmhg)	DIASTOLIC (mmhg)
NORMAL	< 120	< 80
PREHYPERTENSION	120- 139	80-89
HYPERTENSION	>140	>90

Table 2 showing categories of hypertension

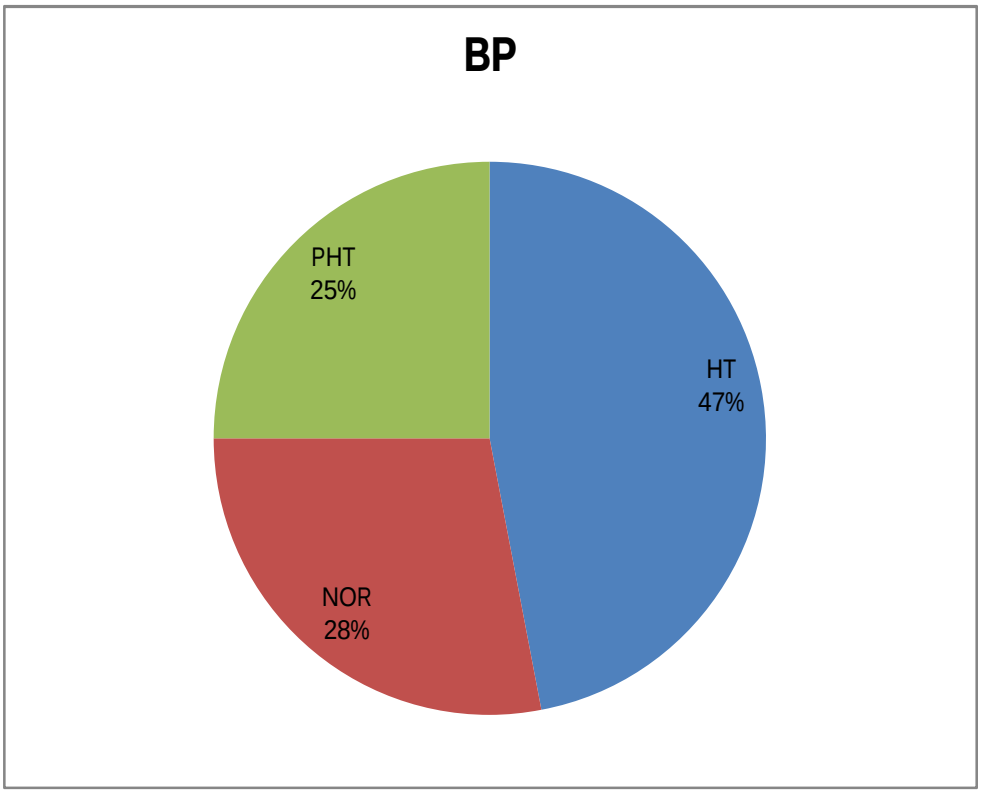

Chart 7 showing Distribution of Hypertension

In this study, out of 100 patients, 25 patients (25%) were in prehypertensive stage. 47 patients (47%) had hypertension.

	NO OF PATIENTS	PERCENTAGE
NORMAL	28	28
PHT	25	25
HT	47	47
TOTAL	100	100

Table 3 showing Distribution of Hypertension

SEVERITY OF GLAUCOMA IN HYPERTENSIVES

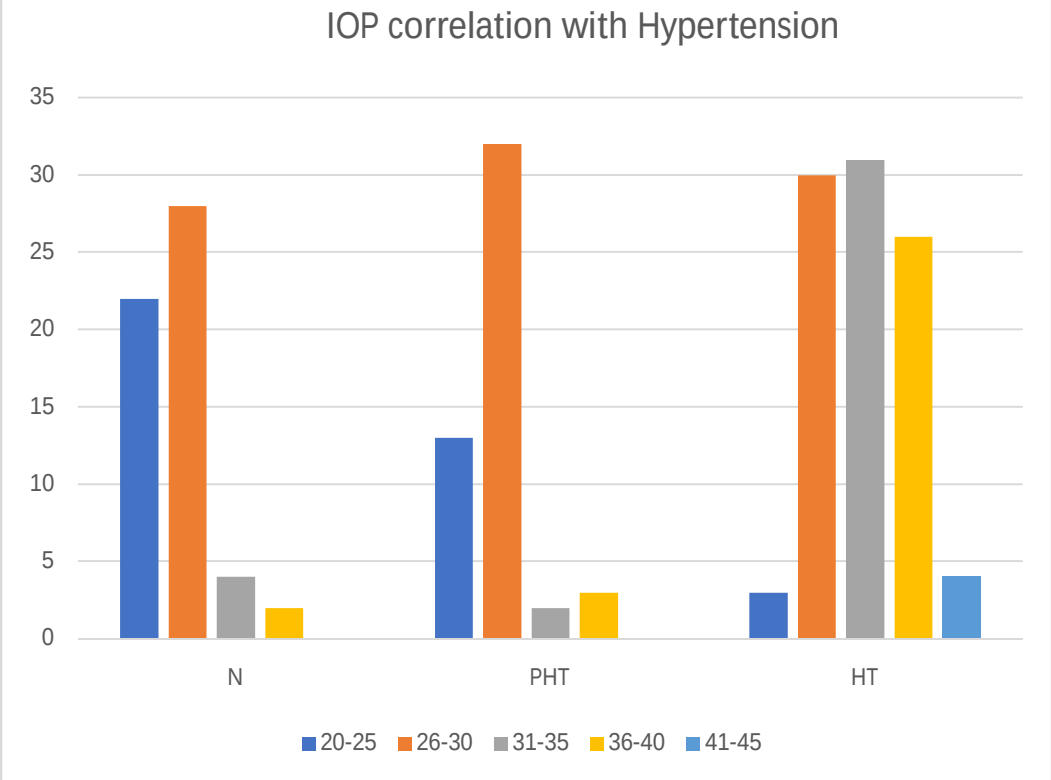

Chart 8 showing HT & IOP correlation

IOP CORRELATION WITH HYPERTENSION

IOP	N	PHT	HT
20-25	22	13	3
26-30	28	32	30
31-35	4	2	31
36-40	2	3	26
41-45	0	0	4

Table 4 showing distribution of IOP in hypertension

In our study, patients without hypertension group had IOP range of 20-30 mmhg. Prehypertensive group had predominantly presented with the IOP range of 26-30 mmhg.

In Hypertensive group, 30% eyes had IOP range of 26-30mmhg. 31% had presented with IOP range of 31-35 mmhg. 26% had presented with iop range of 36-40 mmhg. 4% eyes had IOP range of >40 mmhg.

This observation is statistically significant with p value of 0.000, by chi square analysis.

CDR CORRELATION WITH HYPERTENSION

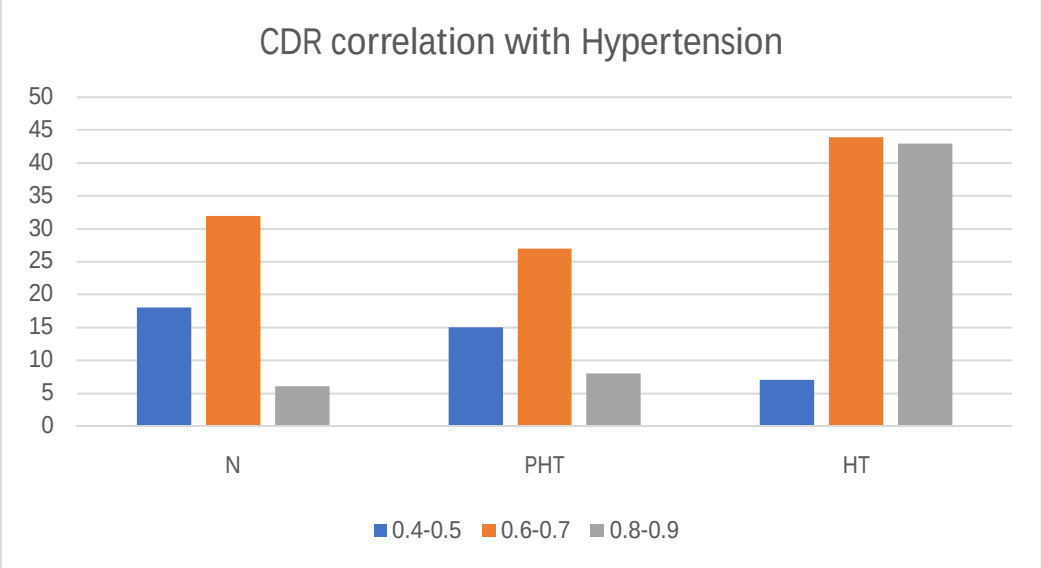

Chart 9 showing distribution of CDR in hypertensive patients

CDR	N	PHT	HT
0.4-0.5	18	15	7
0.6-0.7	32	27	44
0.8-0.9	6	8	43

Table 5 showing distribution of CDR in hypertensives

In our study, patients without hypertension group had presented mostly with CDR range of 0.6-0.7. prehypertensive group had predominantly CDR range of 0.6-0.7.

In Hypertensive group, 43% eyes had CDR range of 0.8-0.9, 44% were having CDR range of 0.6-0.7, 7% eyes had 0.4-0.5.

This observation is statistically significant with p value of 0.000 by chi square analysis.

DIABETES MELLITUS AND GLAUCOMA:

RANDOM BLOOD SUGAR	CATEGORY
< 140 mg/dl	NORMAL
140-199	PREDIABETES
>200	DIABETES

Table 6 showing categories of diabetes

DISTRIBUTION OF DIABETES:

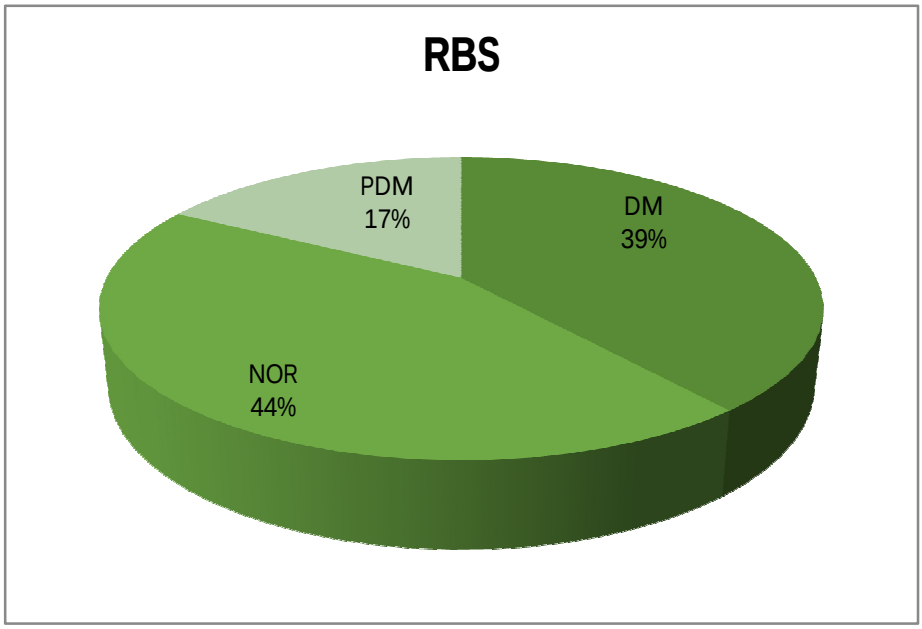

Chart10 showing Distribution of Diabetes.

	NO OF PATIENTS	PERCENTAGE
NORMAL	44	44
PREDIABETTIC	17	17
DIABETIC	39	39
TOTAL	200	100

Table 6 showing Distribution of Diabetes.

In this study, out of 100 patients,

34 patients (17%) were in prediabetic stage,

39 patients (39%) were having diabetes.

IOP Correlation with Diabetes Mellitus

IOP	N	PDM	DM
20-25	21	9	8
26-30	45	15	30
31-35	12	4	21
36-40	9	6	16
41-45	1	0	3

Table 8 showing distribution of IOP in Diabetes.

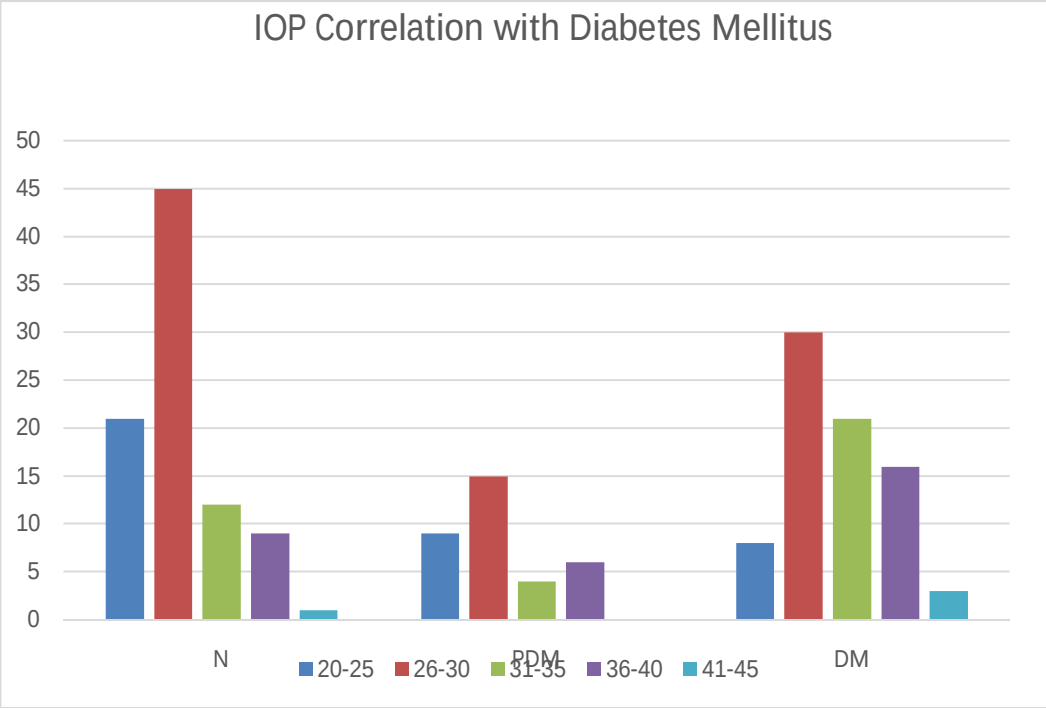

Chart 11 showing distribution of IOP in Diabetes

In this study, patients without diabetes group (88%),most of the patients(66%) were presented with the IOP range of 20-30 mmhg.

Prediabetic group (36%), most of the patients (26%) were in the IOP range of 26-30 mmhg.

In diabetic group (78%), 38% were having below 30mmhg. 40% were having > 30 mmhg.

This observation is statistically significant with p value of 0.031, by chi square analysis.

CDR CORRELATION WITH DIABETES MELLITUS

CDR	N	PDM	DM
0.4-0.5	22	8	10
0.6-0.7	50	18	35
0.8-0.9	16	8	33

Table 9 showing distribution of CDR in diabetes

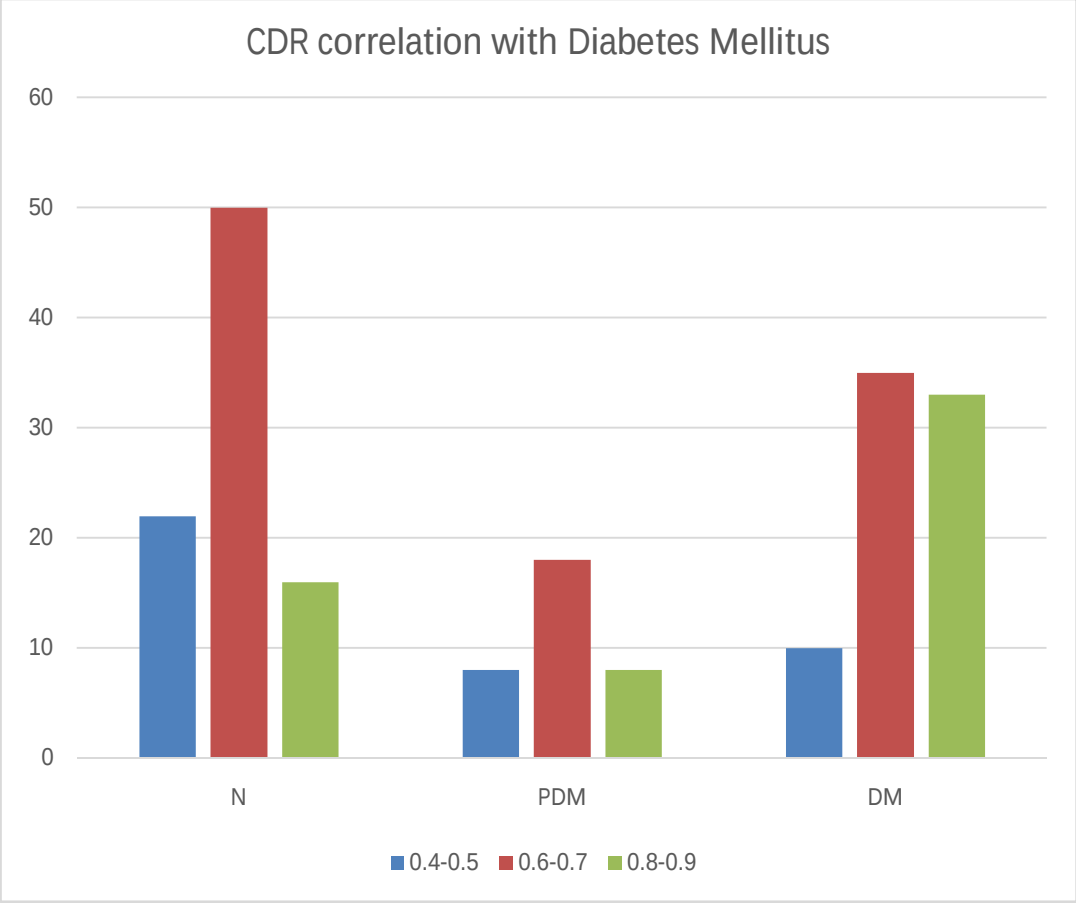

Chart 12 showing distribution of CDR in diabetes.

In patients without diabetes group, most of the patients (50%) were having CDR of 0.6-0.7.

In prediabetic group, 18% were having CDR of 0.6-0.7.

In diabetic group, 35% were having CDR of0.6-0.7, 33% were having CDR of 0.8-0.9.

This observation is statistically significant with p value of 0.031 by chi square analysis.

GLAUCOMA AND CHOLESTEROL ASSOCIATION

DISTRIBUTION OF CHOLESTEROL:

	NORMAL mg/dl	BODERLINE mg/dl	HIGH mg/dl
CHOLESTEROL	< 200	200-239	>240
TRIGLYCERIDES	<150	150-199	>200
LDL	60	35-59	<35
HDL	60-130	130-159	>160

Table 10 showing categories of Cholesterol

	NUMBER OF PATIENTS	PERCENTAGE
NORMAL	53	53
BODERLINE	23	23
HIGH	24	24

Table 10 showing distribution of Cholesterol

In this study, out of 100 patients 23 patients (23%) were having high cholesterol levels,

24 patients (24%) were having borderline cholesterol levels.

CORRELATION OF CHOLESTEROL WITH IOP:

IOP	N	B	H
20-25	21	12	5
26-30	46	20	24
31-35	20	7	10
36-40	15	7	9
41-45	4	0	0

Table 12 showing distribution of IOP in dyslipidemia patients

IOP & DYSLIPIDEMIA

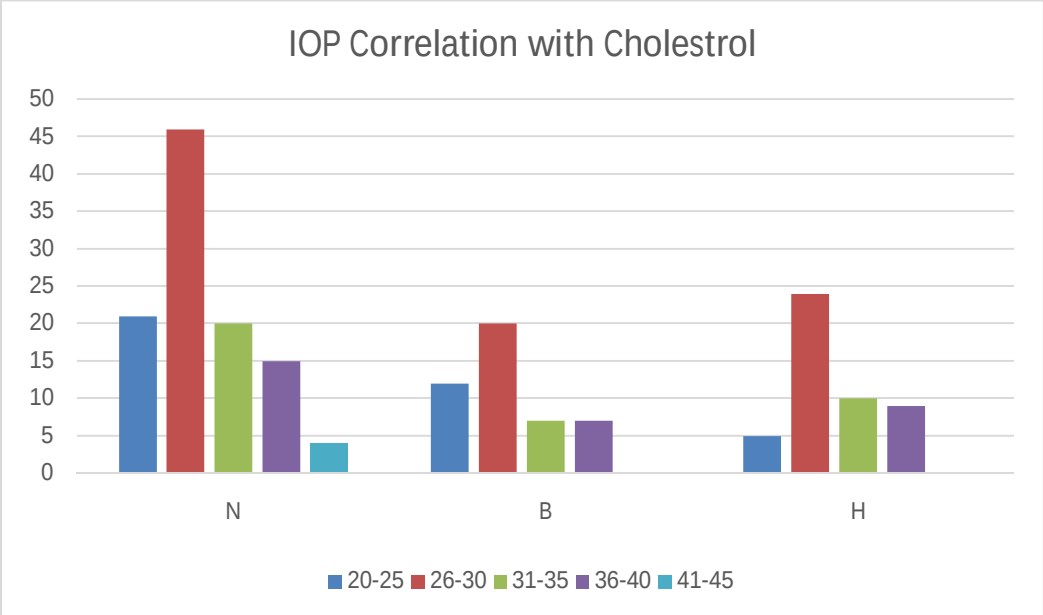

Chart 13 showing correlation of IOP with Cholesterol

In patients without cholesterol group, maximum percentage (86%) were having IOP range of < 35 mmhg.

In borderline group, 32% were having iop range < 30 mmhg.

In high cholesterol group, maximum percentage of patients (34%) were having IOP range of 25-35 mmhg.

So in this study, there was no association of cholesterol with IOP.

CORRELATION OF CHOLESTEROL WITH CDR:

CDR	N	B	H
0.4-0.5	22	11	7
0.6-0.7	46	25	32
0.8-0.9	38	10	9

Table 13 showing distribution of CDR in cholesterol patients

CDR & DYSLIPIDEMIA

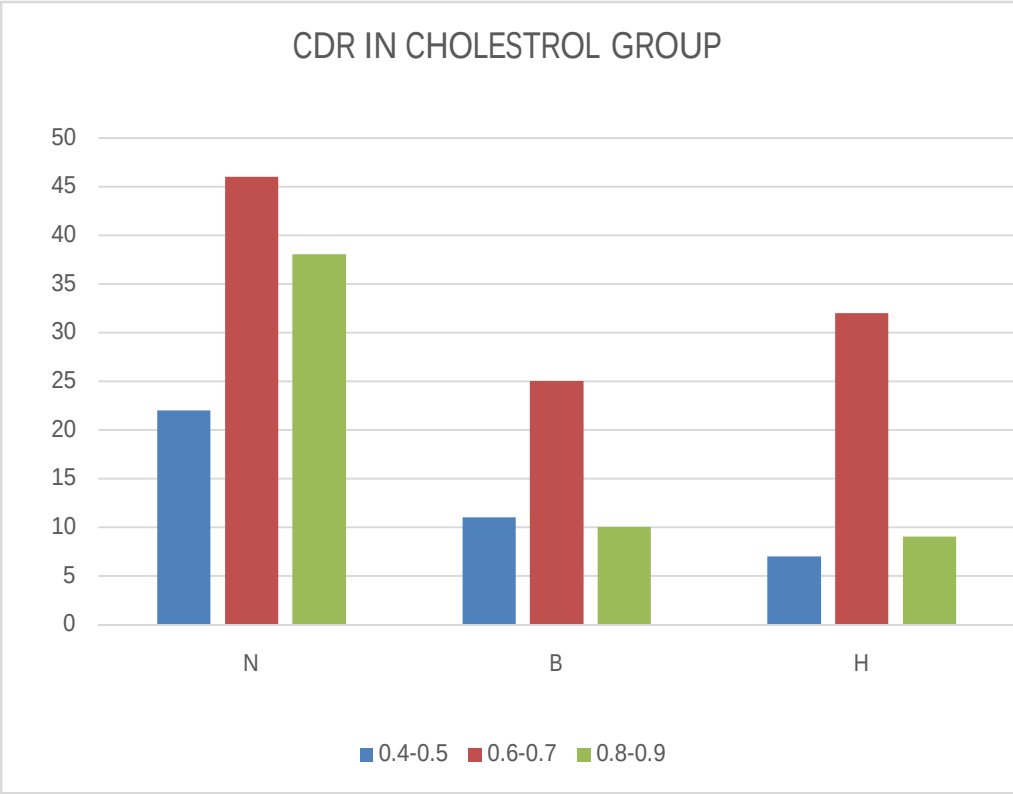

Chart 14 showing distribution of CDR in cholesterol patients

In patients with normal cholesterol levels, maximum percentage (22%) were having CDR range of 0.4-0.5.

In patients with borderline cholesterol levels, 43% had CDR range of 0.6-0.7, 35% had CDR 0.8 - 0.9.

In patients with high cholesterol levels, 67% were having CDR range of 0.6-0.7, 19% had CDR 0.8 - 0.9.

This observation is statistically significant with p value of 0.006 by chi square analysis.

MEAN OCULAR PERFUSION PRESSURE AND GLAUCOMA

It is calculated by 2/3(mean arterial pressure – IOP).

Mean arterial pressure is calculated by Diastolic BP + 1/3

(Systolic BP- Diastolic BP)

MEAN OCULAR PERFUSION PRESSURE DISTRIBUTION:

MOPP	NO OF EYES
30-40	39
41-50	137
51-60	24

Table showing Distribution of MOPP

MOPP WITH IOP CORRELATION:

MOPP	20-30	31-40	>40
30-40	18	17	4
41-50	87	50	-
51-60	23	1	-

Table 15 showing correlation of IOP & MOPP

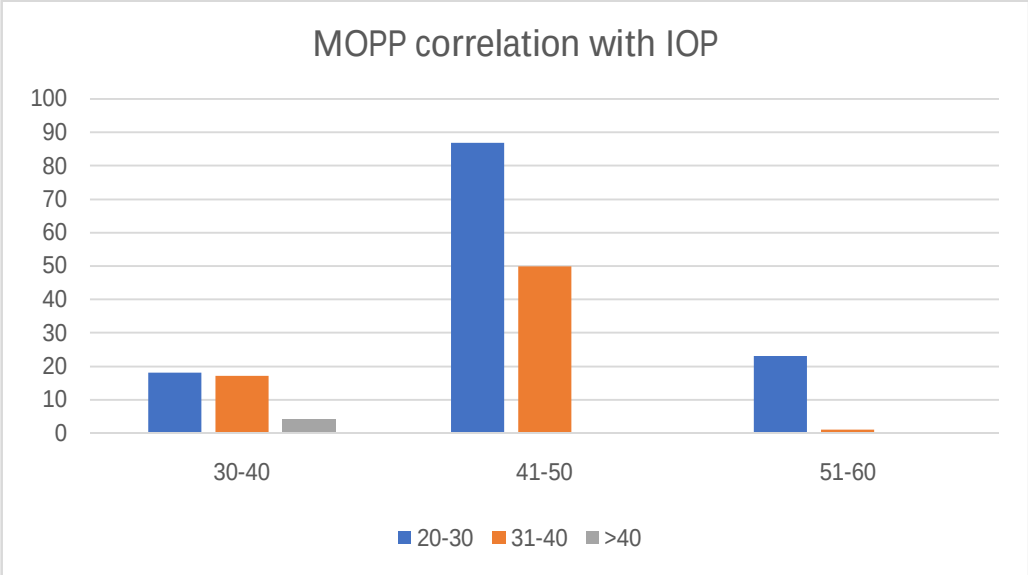

Chart 15 showing correlation of IOP & MOPP

In this study, MOPP 30-40 mmhg group, out of 39 eyes, 18 eyes(46%) had IOP within 20-30 mmhg, 17 eyes(43%) had IOP range of 31-40 mmhg, 4(10%) eyes had IOP > 40 mmhg.

In MOPP 41-50 mmhg group, 87 eyes(63%) had IOP range of 20-30 mmhg, 50 eyes(36%) had IOP range of 31-40mmhg.

In MOPP 51-60 mmhg, 23 eyes(95%) had IOP range of 20-30 mmhg, 1eye(4%) had iop 31-40 mmhg.

Thus we observed a negtive correlation between MOPP and IOP**.**

This observation is statistically significant with p value 0.000 by pearson correlation

MOPP WITH CDR CORRELATION:

MOPP	0.4-0.5	0.6-0.7	0.8-0.9
30-40	-	20	19
41-50	64	36	37
51-60	19	4	1

Table 16 showing correlation of MOPP & CDR

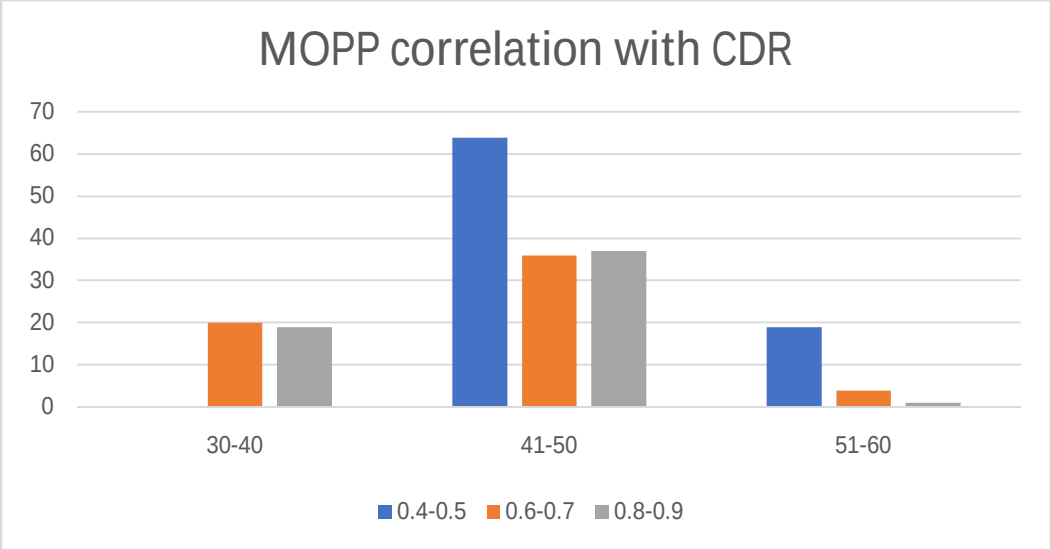

Chart 16 showing correlation of MOPP & CDR

In this study, MOPP 30-40 mmhg group, 51% had CDR 0.6-0.7. 19 eyes (48%) had CDR 0.8-0.9.

In MOPP 41-50 mmhg group, 46% had CDR 0.4-0.5, 26% had 0.6-0.7, 27% had 0.8-0.9.

In MOPP 51-60 group, 79% had CDR 0.4-0.5, 16% had 0.6-0.7, 4% had 0.8-0.9.

We found a negative correlation between MOPP and CDR progression. **This observation is statistically significant with p value of 0.000 br pearson analysis**

DISTRIBUTION OF OTHER DISEASES:

	NO OF PATIENTS	PERCENTAGE
NORMAL	87	87
ANEMIA	13	13

Table 17 showing distribution of Anemia.

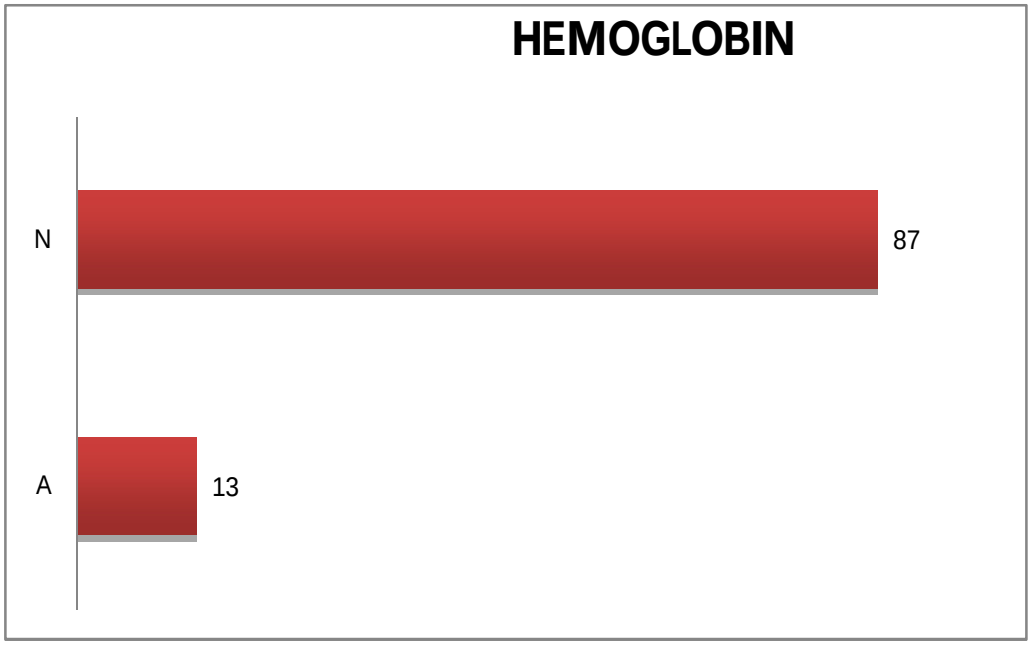

Chart 17 showing Distribution of Anemia

In this study, out of 100 patients, 13 patients (13%) were having anaemia. Hemoglobin level < 12g/dl is defined as anemia.

THYROID ASSOCIATION AND GLAUCOMA:

	NO OF PATIENTS	PERCENTAGE
NORMAL	88	88
HYPOTHYROID	12	12

Table 18 showing distribution of Thyroid disease

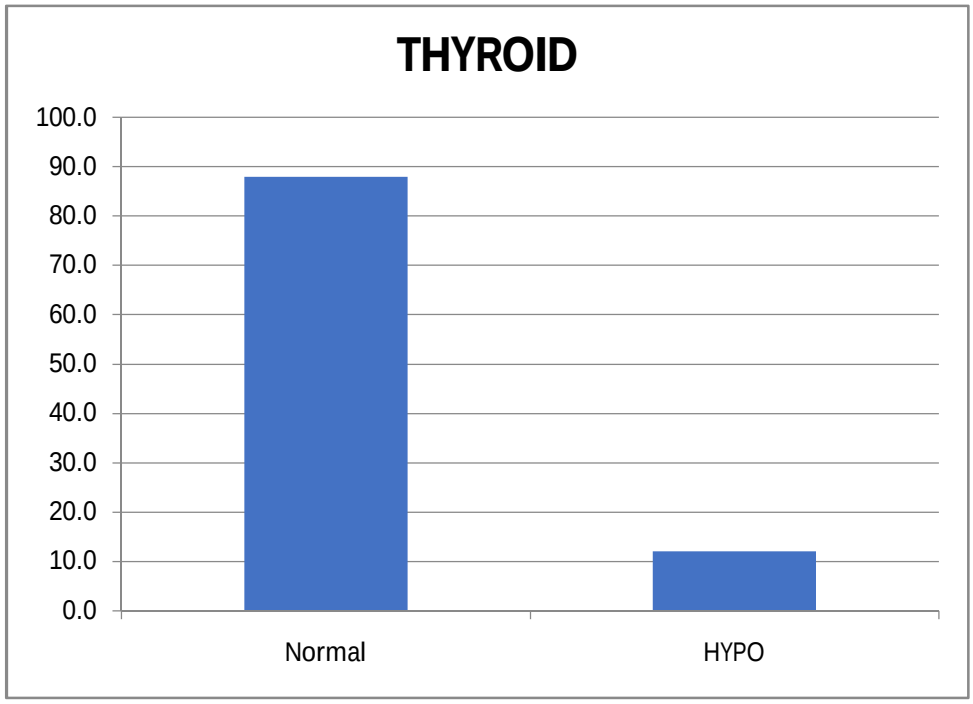

Chart 18 showing distribution of Thyroid

12 patients (12%) were having hypothyroid.

ASSOCIATION OF CARDIAC DISEASE AND GLAUCOMA:

ECG	NO OF PATIENTS	PERCENTAGE
NORMAL	93	93
ABNORMAL	7	7

Table 19 showing distribution of Cardiac disease

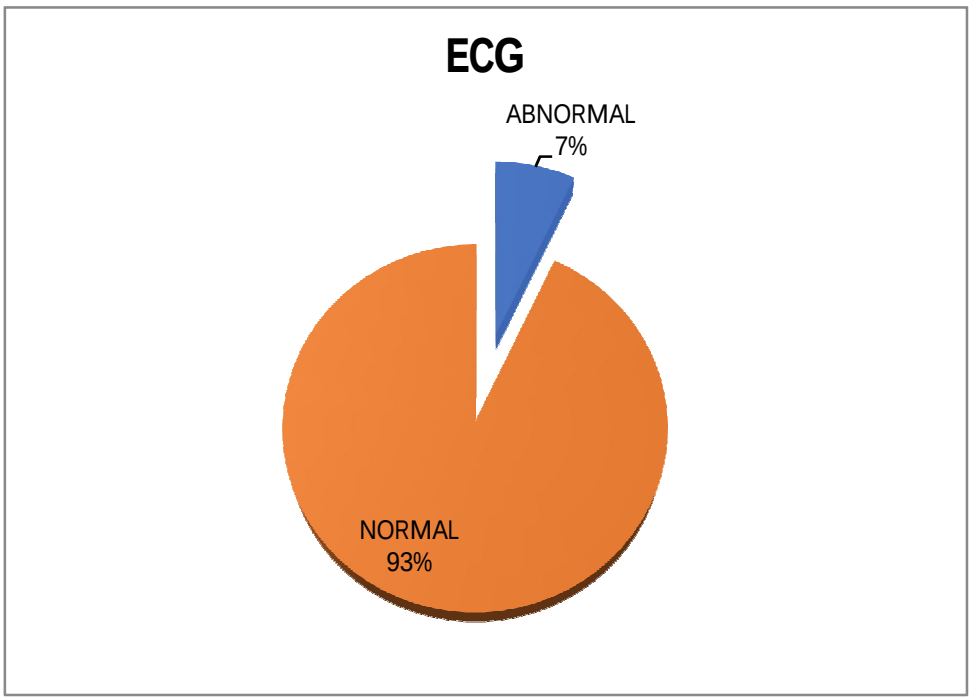

Chart 19 showing distribution of Cardiac disease

7 patients (7%) were showing changes in ECG, which includes left ventricular hypertrophy, coronary heart disease.

DISTRIBUTION OF SYSTEMIC ASSOCIATIONS:

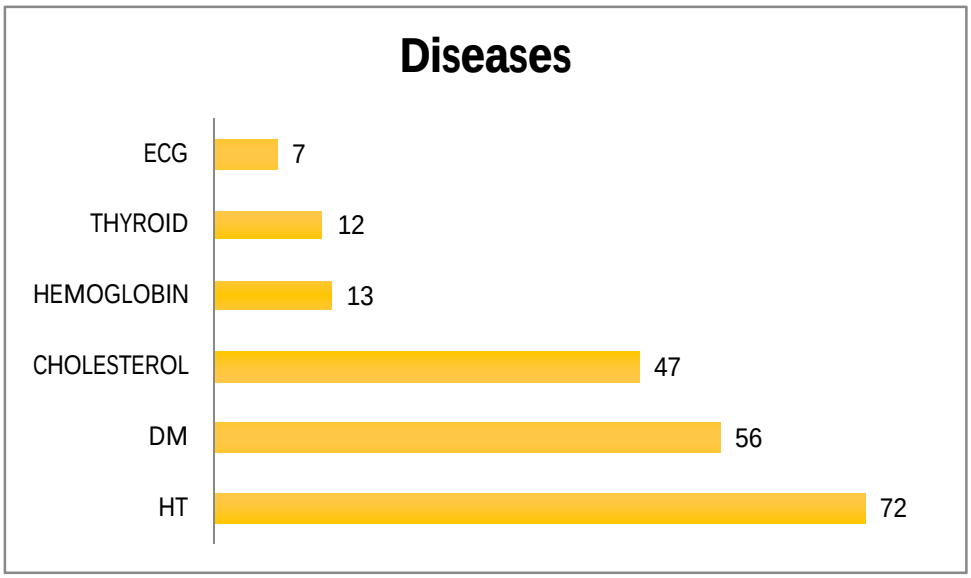

Chart 20 showing distribution of Systemic disease

RESULTS

In our study, the commonest age group was between 50-60 years.

Males are slightly more than the females.

In this study, 147 eyes (73.5%) presented with visual acuity of 6/36 or better. 52eyes (26%) presented with visual acuity in the range of 6/60 to HM. The reason for defective vision was refractive error (22%), cataractous lens changes (44%), posterior capsule opacity (3.8%), advanced glaucoma damage (30%).

128 eyes (64%) presented with intraocular pressure in the range of 20-30 mmhg. 68eyes (34%) were presented in the range of 30-40 mmhg. 4eyes (2%) had >40 mmhg.

104eyes (52%) were presented with CDR 0.7- 0.9, 96 eyes (48%) had CDR 0.4-0.6.

124 eyes (62%) had nasalisation of vessels, 112 eyes (56%) had bayonetting of vessels, 132 eyes (66%) had laminar dot sign, 44 eyes (22%) had barring of circumlinear vessels, 116 eyes (58%) were presented with focal notching, 62 eyes (31%) had parapapillary atrophy

140eyes (70%) were having both superior and inferior arcuate scotomas. 10eyes (5%) were presented with tubular field of vision.

On systemic evaluation, 25 patients (25%) were in prehypertension stage. 47patients (47%) were hypertensives. Prehypertensives were having IOP range of 26-30mmhg and CDR range of 0.6-0.7. Hypertensive group, 61% were having IOP range of

>30mmhg, 43% were having CDR range of0.8-0.9,44% were having CDR of 0.6-0.7.

17 patients (17%) were in prediabetic stage, 39patients (39%) were diabetics. Prediabetic group were having IOP range of 26-30 mmhg and CDR range of 0.6-0.7. Diabetic group were having IOP range of >30 mmhg, 35% having CDR range of 0.6-0.7 and 33% having 0.8-0.9.

23 patients were having cholesterol in the borderline.24 patients were having high cholesterol levels. Borderline cholesterol group, most of the patients (70%) were having IOP range of <30 mmhg, CDR range of 0.6-0.7. High cholesterol group were having IOP range of 25-35 mmhg, 67% patients were in CDR range of 0.6-0.7, 19% having 0.8-0.9.

39 patients were having MOPP of <40 mmhg. In this group, 53% had IOP>30mmhg. 51% had CDR 0.6-0.7, 48% had CDR 0.8-0.9.

7 patients (7%) were showing ECG changes which includes left ventricular hypertrophy and.13 patients (13%) were anaemic. 12 patients (12%) were presented with hypothyroidism.

Out of 100 patients, 87 patients had systemic diseases. Among them maximum number of patients (47%) had hypertension. The second most common systemic disease presented was diabetes (39%).

DISCUSSION

100 patients with primary open angle glaucoma who satisfied the eligibility criteria were enrolled for this study. They were evaluated with proper history taking, visual acuity examination, anterior segment examination by slit lamp, IOP measurement, detailed fundus examination, visual field test by automated perimetry. All the patients have undergone blood pressure measurement, random blood sugar examination, lipid profile, thyroid profile, hemoglobin level, ECG changes.

In this study, the most common age group was 50-70 years. This indicates that POAG is a disease of elderly. Males were slightly more affected than females.

In our study, maximum number of patients were presented with cup disc ratio of 0.8 – 0.9 and tubular field of vision. This indicates the asymptomatic nature of the disease. We also found that maximum number of eyes had superior and inferior arcuate scotomas which correlated well with the advanced cupping and 5% patients presented with tubular field of vision. These were absolute field defects.

Ocular blood flow is determined by mean ocular perfusion pressure. So decrease in perfusion pressure compromises blood flow to ocular region, if there is no autoregulation.

Hypertension and Diabetes which are microangiopathic diseases leading to vascular compromise to optic nerve head leading to glaucoma.

Dyslipidemia causes atherosclerosis of vessels, resulting in vascular compromise to optic nerve head.

cristina leske suggested in their study that low ocular perfusion pressure was significantly associated with open angle glaucoma and also established a positive correlation between blood pressure and intraocular pressure.

Mitchell, Paul, Lee, Anne et al suggested that poorly controlled hypertension is related to increased risk of glaucoma.

T Sato, S Roy. suggested in their study that aqueous humor of diabetic patients show increased glucose which increases fibronectin synthesis and accumulation in trabecular cells resulting in failure of aqueous outflow facility. They also suggested a common biological link between vascular endotheial cells and trabecular cells due to high glucose levels.

Suzana pavljasevic suggeted in their study that dyslipidemia is one of the important risk factor in the pathogenesis of POAG. They also suggested that hyperlipidemia is highly atherogenic and low antioxidant activity. Treatment with statins could reduce the risk of glaucoma appearance.

CONCLUSION

This study indicates that there is a significant association between primary open angle glaucoma and hypertension, diabetes and dyslipidemia.

There is a negative correlation between mean ocular perfusion pressure and severity of glaucoma.

This study results correlates well with previously done studies in systemic factors association in POAG. So this study results highlights the role of vascular factors in the pathogenesis of POAG.

It also emphasizes that all primary open angle glaucoma patients should undergo systemic investigations to find out associated hypertension, diabetes and hypercholesterolemia thereby preventing the further progression of glaucoma.

This study also indicates the importance of a good interaction between ophthalmologist and physician, so that patients showing vascular insufficiency and atherosclerosis due to diabetes, hypertension and high cholesterol levels should be screened for POAG also. Thereby we can diagnose the primary open angle glaucoma at an early stage before significant loss of retinal ganglion cells and further blindness can also be prevented.

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CLINICAL STUDY OF ASSOCIATION OF SYSTEMIC FACTORS IN PRIMARY OPEN ANGLE GLAUCOMA IN A TERTIARY CARE CENTER

PROFORMA

CASE NUMBER:

1. Name: Age / Sex: Mobile No:

OP No/Date:

Place:

2. Symptoms: (Duration)

Defective vision Pain Redness Photophobia

Any other symptoms:

3. Past history -

History of any intraocularsurgery / trauma / Laser

4. Medical history –

Diabetes / hypertension / IHD / vaso occlusive disease / bronchial asthma / chronic disease / steroid usage (long term)/ Dyslipidemia/ Anemia

5. Family history-

History of diabetes / hypertension / ischemic heart disease / vein occlusion diseases in the family

6. Ocular Examination:

Vision: (BCVA)

RE: LE:

Tension:

GAT/Rebound tonometry: RE: mmhg LE: mmhg

	RE	LE
Lids		
Conjunctiva		
Cornea		
Anterior chamber: Depth Cells/flare		
Iris		
Pupil: Size shape reaction		
Lens :		

Gonioscopy: schaffer RE  LE 

Fundus : RE: media: disc and vessels:

LE: media: disc and vessels:

7. Other Investigations:

8. Diagnosis:

9.Treatment Plan:

Medical: Topical drugs

Surgical: Trabeculectomy / anti glaucoma device / Laser trabeculoplasty

10. Follow up: Monthly Visits

Visual acuity

Anterior segment

Fundus

IOP

Gonioscopy

Fields

KEY TO MASTER CHART

IP/NO	-	INPATIENT NUMBER
CDR	-	CUP DISC RATIO
IOP	-	INTRAOCULAR PRESSURE
AP	-	AUTOMATED PERIMETRY
RE	-	RIGHT EYE
LE	-	LEFT EYE
A	-	ABSOLUTE DEFECT
R	-	RELATIVE DEFECT
S	-	SUPERIOR ARCUATE AREA
I	-	INFERIOR ARCUATE AREA
NS	-	NASAL STEP
PC	-	PARACENTRAL
TUB	-	CENTRAL TUBULAR FIELD OF VISION
NP	-	NOT POSSIBLE
HT	-	HYPERTENSION
PHT	-	PREHYPERTENSION
DM	-	DIABETES
PDM	-	PREDIABETIC
N	-	NORMAL
B	-	BORDER LINE
H	-	HIGH
A	-	ANEMIA
HYPO	-	HYPOTHYROID
AB	-	ABNORMAL
MOPP	-	MEAN OCULAR PERFUSION PRESSURE

MASTER CHART

NO	NAME	AGE	SEX	IP NO	VISION		IOP		CDR		AP		BP	RBS	CHOLESTEROL	HEMOGLOBI	THYROID	ECG	MOPP	
					RE	LE	RE	LE	RE	LE	RE	LE							RE	LE
1	Thora	60	F	4365	6/12	6/18	22	26	0.5	0.6	R/S	R/S	HT	N	H	N	N	N	58	54
2	Ellamal	50	M	1191	6/36	6/60	34	30	0.9	0.8	TUB	A/S/I	HT	DM	N	N	HYPO	N	46	50
3	Deenadayalan	55	M	1595	6/18	6/36	22	24	0.4	0.5	R/S/I	R/S/I	PHT	DM	B	A	N	N	48	46
4	jayalakshmi	48	F	1734	6/24	6/18	30	30	0.6	0.6	R/S/I	R/S/I	N	PDM	N	N	N	N	48	50
5	Rajagopal	61	M	1737	4/60	6/60	34	36	0.9	0.9	NP	TUB	HT	DM	N	N	N	N	46	44
6	Selvaraj	63	M	5309	6/60	4/60	26	32	0.7	0.9	A/S/I	NP	HT	DM	B	N	N	AB	54	48
7	Pandiyan	50	M	3793	5/60	6/60	34	30	0.9	0.8	NP	A/S/I	PHT	DM	N	N	N	N	36	40
8	Santhanalakshmi	59	F	7943	6/12	6/18	26	28	0.5	0.6	R/S/I	R/S/I	HT	N	H	N	N	N	54	52
9	Saravan	46	M	1778	6/60	4/60	34	32	0.9	0.9	TUB	NP	HT	PDM	N	N	N	N	46	48
10	Suguna	49	F	1107	6/12	6/18	22	26	0.6	0.7	R/S/I	A/S/I	N	PDM	N	N	N	N	46	42
11	Sarathy	64	M	1933	6/36	5/60	34	36	0.8	0.9	A/S/I	NP	HT	DM	N	N	N	N	46	44
12	Ramachandran	57	M	1679	6/18	6/18	28	26	0.7	0.6	R/S/I	R/S/I	PHT	DM	H	N	N	N	42	44
13	Shanmugam	67	M	3374	6/36	3/60	30	34	0.8	0.9	A/S/I	NP	HT	N	N	N	N	N	50	46
14	Chakarai	70	M	4172	6/12	6/12	28	24	0.6	0.5	R/S/I	R/S/I	HT	DM	N	N	N	AB	52	56
15	ponnamal	42	F	7899	6/60	6/36	32	26	0.9	0.7	TUB	A/S/I	HT	PDM	N	N	N	N	48	54
16	Manonmani	49	F	4213	6/12	6/18	22	24	0.5	0.6	R/S/I	R/S/I	PHT	DM	H	A	HYPO	N	48	46
17	Pandiyan	65	M	5348	6/18	6/24	30	34	0.4	0.6	R/S/I	R/S/I	HT	DM	B	N	N	N	50	46
18	Tamilarasi	56	F	1779	6/24	6/36	26	24	0.7	0.6	A/S/I	R/S/I	N	PDM	N	N	N	N	42	44
19	Arputharaj	44	F	1571	4/60	6/36	34	36	0.9	0.8	NP	A/S/I	N	N	N	N	N	N	34	32
20	Jeyanathan	69	M	1762	6/36	6/12	26	22	0.6	0.5	R/S/I	PC	PHT	N	B	N	N	N	44	48
21	Anjan	54	M	1518	6/24	6/36	32	28	0.5	0.6	R/S/I	R/S/I	HT	DM	H	N	N	AB	48	52
22	Sakunthala	46	F	4418	3/60	6/36	26	28	0.9	0.7	NP	A/S/I	N	DM	N	A	HYPO	N	42	40
23	Venkat	49	M	9331	2/60	6/24	32	28	0.8	0.7	NP	A/S/I	HT	PDM	N	N	N	N	48	52
24	Malka	57	F	5341	6/60	6/36	30	28	0.6	0.7	A/S/I	A/S/I	HT	DM	B	N	N	N	50	52
25	Govind	63	M	3308	6/36	4/60	30	36	0.7	0.9	A/S/I	NP	HT	DM	H	N	N	N	50	44
26	Kailasam	62	M	1906	6/24	6/18	30	26	0.7	0.6	R/S/I	R/S/I	HT	DM	H	N	N	N	50	54
27	Jayalakshmi	48	F	1734	6/12	6/18	22	24	0.5	0.5	R/S/I	R/S/I	N	N	N	N	HYPO	N	46	44
28	Elumalai	57	M	1291	6/60	6/24	38	32	0.8	0.6	A/S/I	R/S/I	HT	PDM	B	N	N	N	42	48
29	Veerasamy	52	M	1001	6/18	6/24	28	30	0.6	0.6	R/S/I	R/S/I	PHT	N	N	N	N	N	42	40
30	Rajamani	49	M	1778	6/36	6/12	24	22	0.7	0.5	A/S/I	NS	N	DM	B	N	N	N	44	46
31	Ettiyappan	62	M	1720	6/36	6/60	36	34	0.8	0.8	A/S/I	A/S/I	HT	DM	H	N	N	N	44	46
32	Sivagami	54	F	1793	6/18	6/36	26	28	0.6	0.7	A/S	A/S/I	PHT	N	N	N	N	N	44	42
33	Selvaraj	57	M	1878	6/36	6/24	40	36	0.8	0.7	A/S/I	A/S/I	HT	PDM	N	N	N	N	40	44
34	Geetha	51	F	1534	6/18	6/24	24	26	0.5	0.6	NS	R/S/I	PHT	N	B	N	N	N	46	44
35	Govindan	68	M	1614	6/60	6/36	42	40	0.9	0.8	TUB	A/S/I	HT	DM	N	N	N	N	38	40
36	Kokilam	59	F	1252	6/36	6/24	30	34	0.7	0.7	A/S/I	A/S/I	HT	DM	H	N	N	N	50	46
37	Manickam	63	M	1355	6/12	6/36	28	36	0.6	0.8	R/S/I	A/S/I	HT	N	N	N	N	N	52	44
38	Munusamy	69	M	1576	1/60	6/36	40	34	0.9	0.7	NP	A/S/I	HT	DM	N	N	N	N	40	46
39	Rajeswari	46	F	1273	6/24	6/12	24	20	0.5	0.5	R/S/I	R/S/I	N	PDM	N	N	N	N	44	48
40	Aslam Bhai	68	M	1780	6/36	6/36	38	32	0.8	0.7	A/S/I	A/S/I	HT	DM	H	N	N	AB	42	48
41	Narayanan	45	M	1794	6/18	6/12	22	22	0.5	0.5	R/S/I	R/I	PHT	PDM	N	N	N	N	48	48
42	Sakunthala	43	F	1548	6/24	6/24	24	28	0.4	0.6	NS	R/S/I	N	N	B	A	HYPO	N	44	40
43	Tamilarasan	48	M	1762	6/18	6/12	28	24	0.6	0.5	R/S/I	R/S/I	N	N	N	N	N	N	40	44
44	Valliyammal	65	F	1421	6/12	6/24	30	26	0.6	0.7	R/S/I	A/S/I	PHT	DM	H	N	N	N	40	44
45	Anjammal	63	F	1236	6/60	6/24	38	34	0.8	0.6	A/S/I	A/S	HT	DM	H	N	N	N	42	48
46	Vaduganathan	62	M	1870	6/12	6/18	28	26	0.6	0.6	R/S/I	R/S/I	PHT	PDM	N	N	N	N	42	44
47	Shenbagam	54	F	1935	6/24	6/12	26	28	0.7	0.6	A/S/I	R/S/I	PHT	DM	N	N	N	N	44	42
48	Santhy	49	F	1569	6/24	6/18	22	24	0.6	0.6	R/S	R/S/I	N	N	B	A	HYPO	N	46	44
49	Kumaresan	44	M	1543	6/24	6/18	24	22	0.5	0.5	NS	R/S/I	N	N	N	N	N	N	44	46
50	Valliyappan	65	M	1446	6/36	6/60	32	38	0.7	0.8	A/S/I	A/S/I	HT	DM	H	N	N	N	48	42
51	KUMARESAN	60	M	4866	6/36	6/60	28	30	0.7	0.7	A/S/I	A/S/I	HT	N	H	N	N	N	52	50
52	ANNADURAI	50	M	4883	6/12	6/24	24	24	0.5	0.6	R/S	R/S	N	N	N	N	N	N	44	44
53	GOVINDHAN	55	M	5156	6/36	6/60	32	34	0.8	0.8	A/S/I	A/S/I	HT	DM	B	N	N	N	48	46
54	ADIKESAVAN	48	M	3337	6/24	6/12	26	24	0.5	0.4	R/S	R/S	N	PDM	N	N	N	N	42	44
55	ESHWARI	45	F	5324	6/36	6/60	34	36	0.8	0.9	A/S/I	T	HT	DM	N	A	HYPO	N	46	44

56	SAMY	63	M	5224	6/18	6/12	26	26	0.4	0.5	NS	R/I	PHT	DM	B	N	N	N	44	44
57	SELVARAJ	50	M	4608	6/60	6/36	28	26	0.8	0.7	A/S/I	A/S/I	PHT	PDM	N	N	N	N	42	44
58	SIVAGAMI	59	F	5185	6/36	6/24	28	26	0.6	0.5	R/S/I	R/I	HT	N	H	N	N	N	52	54
59	ABDUL HAMEED	46	M	7130	6/60	5/60	32	38	0.8	0.8	A/S/I	A/S/I	N	DM	N	N	N	N	36	30
60	VASANTHA	49	F	7835	6/36	6/60	30	28	0.7	0.6	A/S/I	R/S/I	N	N	B	A	N	N	38	40
61	NAGAMMAL	64	F	1909	6/24	6/36	28	28	0.6	0.7	R/S/I	A/S/I	HT	PDM	N	N	HYPO	N	52	52
62	MUNIYANDI	57	M	7131	6/60	5/60	28	30	0.7	0.7	A/S/I	A/S/I	PHT	DM	H	N	N	N	42	40
63	CHINNASAMY	67	M	8603	6/36	6/60	26	26	0.6	0.6	R/S/I	R/S/I	N	N	N	N	N	N	42	42
64	GOVINDASAMY	70	M	6599	6/60	6/36	40	38	0.8	0.8	A/S/I	A/S/I	HT	DM	B	N	N	AB	40	42
65	ABDUL BASHA	42	M	3067	6/24	6/36	24	26	0.5	0.4	R/S	PC	N	PDM	N	N	N	N	44	42
66	NALLU	49	M	5179	HM	1/60	36	38	0.9	0.9	NP	NP	PHT	DM	N	N	N	N	34	32
67	RADHA	65	F	5348	6/36	6/60	28	28	0.6	0.6	R/S/I	R/I	PHT	DM	B	N	N	N	42	42
68	ASHMATH BEEVI	56	F	7262	6/24	6/36	28	28	0.6	0.6	R/S	A/I	N	PDM	N	N	N	N	40	40
69	ARUL	44	M	7693	6/36	5/60	44	32	0.7	0.9	A/S/I	T	HT	DM	N	N	N	N	36	48
70	VIRUTHAMBAL	65	F	1750	6/60	5/60	28	26	0.7	0.8	A/S/I	A/S/I	PHT	N	B	N	N	N	42	44
71	ANJAMMAL	52	F	9139	6/60	6/60	32	32	0.7	0.7	A/S/I	A/S/I	HT	PDM	H	A	N	N	48	48
72	KASTHURI	46	F	9250	6/12	6/24	24	26	0.5	0.5	R/S	R/S	N	DM	N	A	HYPO	N	44	42
73	VENGATESAN	49	M	1568	6/60	5/60	32	32	0.8	0.7	A/S/I	A/S/I	HT	DM	N	N	N	N	48	48
74	LAKSHMI	47	F	4351	6/60	6/36	28	22	0.7	0.7	A/S/I	A/S/I	PHT	DM	B	A	N	N	42	48
75	GOVINDASAMY	63	M	7586	6/24	6/36	26	28	0.6	0.6	R/S	R/S/I	N	N	N	N	N	N	42	40
76	Sunitha	46	F	317355	6/12	6/12	24	26	0.5	0.5	PC	R/S/I	PHT	N	N	N	N	N	44	42
77	Kalaimani	40	M	535381	6/18	6/18	22	24	0.4	0.5	NS	R/S/I	N	N	N	N	N	N	46	44
78	Radha	55	F	533503	6/18	6/24	26	28	0.5	0.7	R/S/I	A/S/I	PHT	N	H	N	HYPO	N	44	42
79	Uma	54	F	179207	6/36	6/18	28	26	0.8	0.6	A/S/I	R/S/I	N	PDM	N	A	N	N	40	42
80	Chennikuzhandhu	60	M	178603	6/18	6/36	38	38	0.6	0.8	R/S/I	A/S/I	HT	DM	B	N	N	N	42	42
81	Lingaprakash	70	M	154629	6/60	6/36	40	36	0.9	0.7	Tub	A/S/I	HT	N	H	N	N	AB	40	44
82	Bhagyalakshmi	58	F	131726	6/12	6/36	30	32	0.6	0.7	R/S	A/S/I	N	DM	N	N	N	N	38	36
83	Parvathi	56	F	51983	6/36	6/12	38	30	0.8	0.6	A/S/I	R/S/I	PHT	PDM	B	N	N	N	32	40
84	Adhikesavan	66	M	535327	6/24	6/36	30	34	0.6	0.7	A/S/I	A/S/I	HT	N	B	N	N	N	50	46
85	Dhilipan	48	M	234616	6/24	6/18	32	28	0.7	0.6	A/S/I	A/S/I	N	N	N	N	N	N	36	40
86	Devaraj	65	M	106330	6/36	6/12	34	28	0.8	0.6	A/S/I	R/I	HT	DM	H	N	N	N	46	52
87	Muniyappan	62	M	534746	6/24	6/60	32	38	0.7	0.8	A/S/I	A/S/I	HT	DM	N	N	N	N	46	52
88	Jagadha	67	F	441952	6/36	6/60	36	42	0.8	0.9	A/S/I	Tub	HT	PDM	N	N	N	N	44	38
89	Kumaresan	58	M	534860	6/18	6/36	28	24	0.6	0.7	A/S	A/S/I	HT	N	H	N	N	N	52	56
90	Shanthi	70	F	45037	HM	6/60	44	38	0.9	0.8	NP	Tub	HT	DM	N	A	N	N	36	42
91	Shyamala	45	F	21756	6/12	6/18	22	24	0.5	0.5	R/S/I	R/S/I	PHT	PDM	B	N	N	N	48	46
92	Chenbagavalli	66	F	530916	6/36	6/12	36	28	0.7	0.6	A/S/I	A/I	HT	PDM	H	N	N	AB	44	52
93	Raman	54	M	514373	6/18	6/12	28	26	0.6	0.6	A/I	R/I	N	DM	N	N	N	N	40	42
94	Soundariya	42	F	405336	6/36	6/9	26	24	0.7	0.5	A/S/I	R/S/I	PHT	N	H	N	HYPO	N	44	46
95	Prabakaran	58	M	179292	6/12	6/24	30	28	0.5	0.6	R/S/I	A/S/I	HT	DM	N	N	N	N	50	52
96	Kousalya	50	F	528009	6/24	6/18	22	26	0.7	0.6	A/S/I	A/S/I	N	DM	N	A	HYPO	N	46	42
97	Kanthamani	68	F	535350	6/36	6/12	38	32	0.8	0.6	A/S/I	R/S/I	HT	N	B	N	N	N	42	48
98	Viruthambal	65	F	535343	6/36	2/60	32	40	0.7	0.9	A/S/I	NP	HT	PDM	H	N	N	N	48	40
99	Pandiyan	50	M	534802	6/36	6/24	28	32	0.8	0.7	A/S/I	A/S/I	PHT	DM	N	N	N	N	42	38
100	Geetha	55	F	179486	6/24	6/60	30	26	0.6	0.7	R/S	A/S/I	N	N	B	N	N	N	38	42